ACTA RADIOLOGICA

EDITA IUSSU SOCIETATUM RADIOLOGICARUM
DANIAE, FENNIAE, NORVEGIAE ET SUECIAE

VOL. 55 JANUARY 1961 FASC. 1

INFLUENCE OF NALORPHINE ON THE SPHINCTER OF ODDI STUDIED BY ROENTGENCINEMATOGRAPHY

by

PERCY LINDGREN and GEORG-FREDRIK SALTZMAN

It has long been known that morphine increases the tone of unstriated intestinal muscle in general and hence that of the sphincter of Oddi as well. Most substances belonging to the morphine group, both natural and synthetic, are considered to exercise a similar influence on unstriated muscle. Nalorphine (N-allylnormorphine) (WINTER et coll. 1954) forms an exception in this respect since it is considered that this substance has an antagonistic effect on the tonic action of morphine (HART and MCCAWLEY 1944).

During the last few years we have studied the influence of various drugs upon the sphincter of Oddi by means of a system consisting of an image intensifier and a film camera adapted for roentgencinematography. Nalorphine has also come in for attention in this connection, and as our results diverge in certain respects from the view generally held we considered they were worth placing on record.

Our knowledge regarding nalorphine as a morphine antagonist is mainly based on experimental and clinical experience with this substance in morphine

From the Department of Pharmacology (Director: Prof. Börje Uvnäs), Karolinska Institutet and the Roentgen Department (Prof. Erik Lindgren), Serafimerlasarettet, Stockholm, Sweden. Submitted for publication 11 July 1960.

intoxication (UNNA 1943, WOODS 1956, FRASER 1957). In general the drug behaves as a narcotic antagonist in the presence of a strong narcotic effect; in the absence of a narcotic effect it behaves as a narcotic, in the presence of narcotic addition it produces withdrawal symptoms. The best known effect of nalorphine is its power to counteract the respiratory depression produced by morphine. The precise mode of action is unknown, although pharmacologic observations suggest that it can best be explained as competitive inhibition.

Nalorphine is said to antagonize intestinal morphine spasm as well as acting as a spasmolytic. HART and MCCAWLEY (1944) observed that in non-anaesthetized dogs nalorphine caused a decrease in tone with a slight increase in activity. When morphine was given first the intestinal tone fell to below the starting level while at the same time a certain degree of hyperactivity was noted. When morphine was administered after nalorphine the morphine spasm was not completely inhibited. Similar results were reported by GRUBER and GRUBER (1953), who found that nalorphine will prevent or antagonize the increase in intestinal tone produced by morphine and the series of drugs of the morphine group.

WINTER et coll. (1954) tested the antispasmodic activity of nalorphine on the isolated ileum of the guinea-pig and rabbit. As compared with papaverine nalorphine has a very weak antispasmodic activity.

Nalorphine has also been reported to produce an inhibition of gastric and intestinal motility in man (BEAL and SCHAPIRO 1953). In another publication the same investigators (SCHAPIRO and BEAL 1953) made a direct study of the effect of nalorphine on the choledochal sphincter action. When nalorphine was given after the increase in intraductal pressure produced by morphine there was a prompt decrease in common duct pressure; when the nalorphine was administered before the morphine, the characteristic rise in intraductal pressure was not produced.

To sum up, it may be said that according to the results reported in the literature nalorphine has a weak, although distinct spasmolytic effect on unstriated intestinal muscle and displays antagonism not only to the central but also to the peripheral effects produced by morphine.

Material and Methods

The effect of nalorphine on the sphincter of Oddi has been studied in 10 cases. The examinations were carried out in connection with postoperative, so-called secondary cholangiographies, in patients who had undergone cholecystectomy about 5 days previously. The 10 cases were taken in unbroken succession, and without selection, from the routine material at Serafimerlasarettet.

The contrast medium used was Hypaque 45 % (sodium diatrizoate), diluted with an equal amount of physiologic salt solution. The medium was injected through a rubber tube which had been introduced into the ductus choledochus at the operation. An amount of 5 ml of contrast medium was given at each injection, the first dose being injected before the nalorphine medication. Immediately after the first injection the patient received 10 mg of nalorphine subcutaneously. Subsequent injections of contrast medium were carried out at 5, 10, 15, and 20 minutes after the nalorphine injection. Using an Arriflex camera adapted for roentgencinematography and connected to a Philips image intensifier the region of the sphincter of Oddi was filmed for 10 to 20 seconds at an exposure rate of 16 films per second immediately after the conclusion of the respective contrast injections. The patient lay in the same position during the whole of the time. Following these film recordings, 2 to 4 drops of nitroglycerine were administered sublingually and the region of the sphincter of Oddi was filmed 1 and 2 minutes after the administration. In addition to these 10 cases a further 3 examinations were carried out in connection with this study. These 3 patients first received 10 mg of morphine subcutaneously and were kept under fluoroscopic observation till a constant contraction had arisen in the sphincter of Oddi; as soon as this had been established they were given 10 mg of nalorphine. Films were then exposed at short intervals for 15 to 20 minutes after the injection of contrast medium.

Results

In none of our cases did the first injection of contrast medium, carried out before the nalorphine injection, cause any demonstrable spasm in the sphincter of Oddi and our experience is thus the opposite of that reported by JACOBSSON et coll. (1959). These investigators held that the sudden pressure increase produced by the injection of contrast medium may provoke a rapidly disappearing or more longstanding increase in the tone of the sphincter muscles. In all of our cases, the medium passed out freely into the duodenum after the first injection, and in all 10 cases the bile ducts were found to be completely free from contrast medium at the fluoroscopic examination preceding the next injection of the medium.

For the assessment of the reaction of the sphincter of Oddi to nalorphine, contractions in the sphincter region were recorded and the widths of the ductus choledochus before and after nalorphine administration were compared. Contraction of the sphincter of Oddi was considered to be present only when no contrast medium could be seen to pass out through the sphincter region into the intestine. Thus, if the lumen of the sphincter was decreased but still allowed visible passage of contrast medium, this was not recorded as a spasm.

In the films taken 5 minutes after nalorphine medication a sphincter spasm that remained unchanged during the entire film recording was demonstrated

a b c

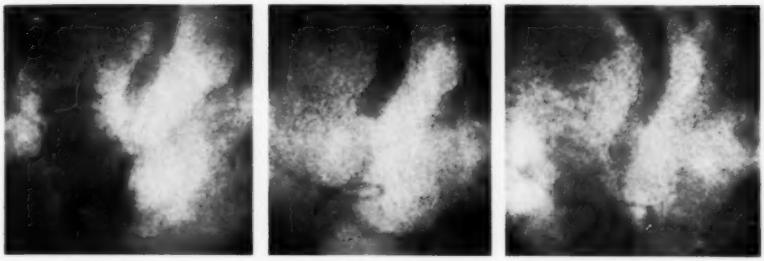
Fig. 1. ♂ 41 years, 75 kg. Cholangiography. Effects of nalorphine on the function of the sphincter of Oddi. a) Before nalorphine. Lower part of common duct contrast-filled. Good passage of contrast medium to the duodenum. b) 10 minutes after 10 mg of nalorphine subcutaneously. Increased tone of the sphincter. No contrast filling of intramural part of the common duct; no passage to the duodenum. c) 25 minutes after nalorphine, 2 minutes after nitroglycerine. The sphincter is relaxed and allows free passage of contrast medium to the duodenum.

in 3 instances; in these cases no contrast medium passed out into the intestine during the 10 to 20 second period represented in the film. In a further 4 cases contractions of short duration, between each of which contrast medium passed into the intestine, were visible; a noticeable change in the relations of the sphincter had, however, taken place in these cases as compared to the appearances in the film taken before nalorphine medication. In the remaining 3 cases no definite contraction of the sphincter could be observed 5 minutes after the injection of nalorphine.

The situation had changed in the films taken 10 minutes after the administration of nalorphine. In all those cases in which transient spasms had been observed after 5 minutes, a contraction that remained constant during the greater part of the film recording was now apparent. A similarly unchanged contraction was also observed in one of the 3 cases in which no spasm had been demonstrated after 5 minutes; in 2 cases there were still no signs of contraction. Of the 3 cases in which a constant contraction was already present after 5 minutes, 2 still showed an unchanged condition after 10 minutes while in the third there were now transient spasms with visible passage of contrast medium in the intervals between them.

With regard to the films taken 15 minutes after injection of the nalorphine it was found that the constant sphincter spasm had relaxed in most of the cases although transient contractions were also now seen in most of them. Rapidly recurring but shortlived contractions were visible in the 2 cases without definite spasm after 10 minutes.

Twenty minutes after the administration of nalorphine the contractions had almost entirely ceased in most of the cases, although in a few instances transient spasms were still visible.



a b c

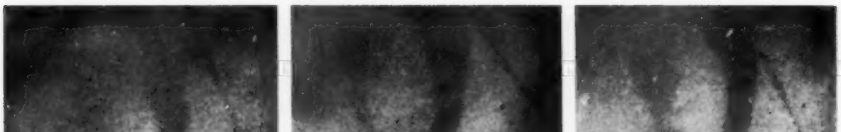
Fig. 2. ♀ 66 years, 60 kg. Cholangiography. Effects of nalorphine on the function of the sphincter of Oddi. Identical conditions and same results as in fig. 1. The concave end of the common duct in (b) indicates a marked spasm in the sphincter and the increased diameter of the duct a higher intraductal pressure.

Summing up it may be said that biliary spasm occurred in all 10 cases. In 8 cases the contraction remained constant for the greater part of at least one of the 10 to 20 second run of films, and in the other 2 cases there were transient spasms that had not been observed before the injection of nalorphine. In most cases the sphincteral contraction reached its maximum 5 to 10 minutes after nalorphine had been given, although the spasms appeared only at a slightly later stage in a few of the cases. The maximal sphincteral contraction lasted for at the most 10 minutes. In a few instances, it was observed at fluoroscopy that the bile ducts had not lost all contrast medium between two film recordings; this was seen only in those cases in which the sphincter spasm had remained constant throughout both film recordings.

It should perhaps be stated that the same dose of nalorphine (10 mg) was administered in all 10 cases. It would, of course, have been more correct to work out the dose in relation to the bodyweight of the patients, and it may therefore be mentioned that the weight in the cases in which no sphincteral contraction lasting throughout the greater part of a film could be observed constituted the heaviest among the patients (80 and 75 kg). The weight of the other patients varied from 47 to 75 kg.

The significance of increased width of the ductus choledochus after morphine medication has been discussed by GUNNARSON (1956) and JONSON (1960) in connection with intravenous cholegraphy. Some difference of opinion has been expressed regarding the diagnostic value of such an increase, but it seems obvious that definite widening must be interpreted as a sign of obstruction to the flow of bile to the intestine. On the other hand, it also seems probable that not every spasm in the sphincter of Oddi need cause widening of the ductus choledochus.

Because of the difficulties experienced in a few cases in obtaining fully satisfactory measurements with the coarse-grained film necessitated by the



a b c

Fig. 3. ♂ 42 years, 67 kg. Cholangiography. Effect of morphine on the sphincter of Oddi. Morphine antagonistic effect of nalorphine. a) Before morphine. Free passage of contrast medium. b) 10 minutes after 10 mg of morphine subcutaneously. Marked spasm of the sphincter. c) 25 minutes after morphine and 10 minutes after 10 mg of nalorphine subcutaneously. Normal conditions seem to have been restored; free passage to the duodenum.

relatively rapid rate of exposure, it was not possible to make exact measurements of the width of the ductus choledochus. Only an unequivocal widening could be recorded as such. Other information may be obtained from Fig. 2. Definite widening of the duct was demonstrated in 7 of the 10 cases; in 2 it was already manifest 5 minutes after the nalorphine injection, in 4 after 10 minutes, and in the seventh 15 minutes afterwards; in no case was such widening seen before a sphincter contraction had been observed. There was no definite evidence of widening in 3 cases. No obvious relationship between widening of the duct and the bodyweight of the patient could be established.

Following nitroglycerine medication, free passage of the contrast medium from the ductus choledochus to the duodenum was observed after 1 minute in every case. In a few instances passage appeared to be free even before the nitroglycerine was given, however. According to our observations the duct in the sphincter region in most instances seemed distinctly wider after nitroglycerine than before the nalorphine medication.

The clinical symptoms and signs could not be recorded in detail in these investigations. Seven of the 10 patients reported that they felt sleepy or dizzy. The size of the pupils was checked in all cases; in 5 of the 10 patients the pupils were much smaller 15 minutes after the nalorphine injection than before it, when examined under the same light conditions.

Sphincter contractions of long duration were rapidly obtained in 2 of the 3 cases in which 10 mg of morphine subcutaneously were given first. In the third case, shortlived but frequent contractions were observed in the sphincter of Oddi.

No sphincter-contracting effect was observed in these cases from the use of nalorphine. Five minutes after the patients had received 10 mg of nalorphine it was no longer possible to distinguish any contraction of the sphincter of Oddi, and the sphincter region was of the same appearance as before the morphine medication.

Although it cannot be asserted with absolute certainty that nalorphine counteracted the action of morphine on the unstriated muscle in these cases, seeing that we are not quite sure when the morphine effect had decreased spontaneously, it is nevertheless clear that the drug did not in these cases cause a contraction of the sphincter such as was demonstrated in all those cases in which nalorphine had been given without preceding morphine medication. It is also obvious that nalorphine did not accentuate the spasmogenic effect of the morphine.

Discussion

Our results, which imply tonic contraction of the sphincter of Oddi after the administration of nalorphine, would appear to contradict the commonly held view regarding the action of this drug on unstriated muscle. The first question that arises is whether our experimental technique may have influenced the result. The injection of contrast medium seems to be the only unphysiologic factor in our examination method. The effect of the pressure increase following the injection has already been mentioned, and it was maintained in that connection that the control injections before the administration of nalorphine did not produce any sphincteral spasm to cause obstruction to the outflow. The contrast medium used, sodium diatrizoate, is relatively pharmacologically inert and can hardly have played any part in producing the sphincter spasm recorded after the nalorphine medication. The film technique also allows recording of the choledochous function directly after the injection of contrast medium.

SCHAPIRO and BEAL (1953), who seem to be the only investigators to have previously studied the effect of nalorphine on the choledochal sphincter in man, examined, as we did, patients who had undergone choledochostomy. They employed a different examination technique, however, and recorded the common duct pressure. The main purpose of their investigation was, however, to study any interaction that might exist between nalorphine and morphine; only in 3 cases were the effects of nalorphine in themselves given attention. According to these authors' curves, 5 mg of nalorphine intravenously did not produce any change, neither an increase nor decrease, in intraductal pressure. It is to be noted, however, that this dose almost completely blocked the action of a subsequent dose of morphine (10 mg intravenously). In the present investigation, 10 mg of nalorphine were given subcutaneously, but whether this was the explanation of the slightly different results is difficult to say. Although our technique did not permit a study of the intraductal pres-

sure it seems obvious to us that a tonic sphincter spasm such as we recorded ought to have produced a rise in pressure. The considerable widening of the common bile duct that we recorded in several instances was in all probability a sign of a pressure increase.

The majority of investigations on the effect of nalorphine on unstriated muscle have not been concerned with the sphincter of Oddi but with the recording of the intraluminal pressure and the peristalsis of other intestinal muscles. Two papers in particular are of interest in this connection; both of them reported decreased intestinal tone after nalorphine. HART and MCCAWLEY (1944) used dogs and gave 0.5 mg/kg intravenously, and BEAL and SCHAPIRO (1953) studied the intestinal motility in man after 5 mg/kg. Although the sphincter of Oddi and other intestinal muscles usually react in a uniform manner to drugs certain observations have suggested that the choledochal sphincter is capable of action independent of the duodenal musculature (SMITH et coll. 1952). For this reason it is difficult to make any comparisons between our results and these earlier observations of the intestinal motility, and they need not necessarily be considered contradictory.

As opposed to our results regarding the auto-effect of nalorphine on the sphincter of Oddi our study of the morphine antagonism has confirmed earlier reports as to a spasmolytic effect (SCHAPIRO and BEAL 1953, ADELMAN and ROSENTHAL 1958, ALPER and VANDAM 1959). Thus, on the one hand nalorphine produces a spasm of the unstriated muscle of the sphincter and on the other has the power to counteract spasm produced by morphine. Before this question can be discussed it would seem necessary to obtain an insight into the effect of nalorphine on other organs and functions.

The best known effect of nalorphine is its capacity in counteracting any respiratory depression produced by morphine. This effect becomes apparent only when the depression is considerable, in other words after the administration of relatively large doses of morphine (FRASER et coll. 1956). In these cases a small dose of nalorphine rapidly restores the respiration to — as far as can be judged — a normal level. On the other hand nalorphine produces in an untreated patient a respiratory depression that may be as severe as that occurring after morphine (FRASER et coll. 1956, HUGGINS et coll. 1957). The same is the case with certain other C. N. S. effects, such as miosis. The explanation of this, according to FRASER et coll., would seem to be a molecular competition between nalorphine and morphine for a cellular receptor site. Both drugs apparently have the same effect, qualitatively speaking, but nalorphine has a considerably greater affinity. Similar opinions as to a competition have been advanced by other authors (e. g. WINTER et coll. 1954).

Our results would seem to indicate a like mode of action with respect to the effect of nalorphine on unstriated muscle. When given alone, nalorphine seems to be bound to certain cell receptors, its effects resembling those of morphine. When nalorphine is administered to patients previously given

morphine, nalorphine competes more successfully than morphine for a site in the cell. The marked sphincter spasm due to morphine might thus be replaced by a weaker tone due to nalorphine, and the effect could be considered as antagonistic. It is possible that smooth muscle needs stronger stimuli to maintain a spasm than to evoke it (an attempt to explain the different effects of the same doses of nalorphine). However, before being taken for granted the hypothesis needs support from further investigations and the establishment of proper dose-response relationships.

Hence, our observations would support the theory that nalorphine has a uniform mode of action, both in its central and peripheral effects, in other words that in all essentials it possesses morphine-like properties but with quantitative differences in its effects; because of a greater affinity for some hypothetical receptor site, nalorphine acquires an apparently morphine-antagonistic action.

To characterize nalorphine as a spasmolytic appears to us immaterial and probably has no applicability in intestinal or biliary spasm. Nor do we share the enthusiasm of ADELMAN and ROSENTHAL (1958) in using nalorphine as 'the agent of choice in the treatment of narcotic-induced spasm'. In the latter cases as well, true spasmolytics such as amyl nitrite, nitroglycerine, and theophylline would appear to be preferable.

Conclusions

1. Nalorphine in a subcutaneous dose of 10 mg produced increased tone of the sphincter of Oddi in all 10 cases examined; marked spasm was demonstrated in 8 cases. The maximum effect was noted after 5 to 10 minutes.

2. The nalorphine spasm was effectively relaxed by 2 to 4 drops of nitroglycerine given sublingually.

3. Nalorphine has a distinctly morphine-antagonistic effect, since 10 mg of nalorphine counteracted and inhibited a spasm in the sphincter of Oddi produced by 10 mg of morphine.

4. The results support the hypothesis that nalorphine and morphine have the same qualitative action, not only on the central nervous system but also on unstriated muscle cells. The tonicizing effect of nalorphine is weaker than that of morphine, but as it probably has a greater affinity for the receptor site it competes favourably with morphine. The result will appear as a relative spasmolysis. This hypothesis is in complete analogy with the current view regarding the mode of action of nalorphine on the central nervous system.

5. Nalorphine should not be termed a spasmolytic. Its practical value as a means of counteracting narcotic-induced intestinal spasm may be said to be inconsiderable.

SUMMARY

The function of the sphincter of Oddi was studied with the aid of roentgencinematography in 10 patients undergoing postoperative cholangiography. A subcutaneous injection of 10 mg nalorphine produced increased sphincter tone (spasm) in every case. This is opposed to certain earlier reports in which spasmolytic properties were attributed to nalorphine. When 10 mg morphine were given first nalorphine counteracted the morphine spasm, as other investigators have also observed.

ZUSAMMENFASSUNG

Die Funktion des sphincter Oddi wurde mit Hilfe der Röntgencinematographie 10 Patienten, die sich einer postoperativen Cholangiographie unterzogen hatten, studiert. Eine sc. Injektion von 10 mg Nalorphin erzeugte in allen Fällen eine Erhöhung des Sphinktertonus (Spasmus). Dies steht im Gegensatz zu gewissen früheren Berichten, in denen dem Nalorphin spasmolytische Eigenschaften zugeschrieben worden waren. Nach Vorbehandlung mit 10 mg Morphin wirkte Nalorphin dem Sphinkterspasmus entgegen wie bereits andere Untersucher beobachtet haben.

RÉSUMÉ

La fonction du sphincter d'Oddi a été étudiée par radiocinématographie chez 10 malades au cours de cholangiographies post-opératoires. L'injection sous-cutanée de 10 mg de nalorphine a augmenté la tonicité du sphincter (spasme) dans tous les cas. Ceci est en contradiction avec certaines publications antérieures, dans lesquelles on attribuait des propriétés spasmolytiques à la nalorphine. Si on a administré d'abord 10 mg de morphine, la nalorphine supprime le spasme morphinique, comme l'ont observé d'autres auteurs.

REFERENCES

ADELMAN, M. H. and ROSENTHAL, A. I.: Nalorphine in the treatment of morphine-induced biliary colic. J. M. Sinai Hosp. 25 (1958), 36.

ALPER, M. H. and VANDAM, L. D.: Morphine, biliary spasm and nalorphine. Anaesthesia 20 (1959), 713.

BEAL, J. M. and SCHAPIRO, H.: Effect of N-allylnormorphine on gastrointestinal motility. Surgery 33 (1953), 65.

FRASER, H. F.: Human pharmacology and clinical uses of nalorphine (N-allylnormorphine). Med. Clin. N. Amer. 41 (1957), 393.

— VAN HORN, G. D. and ISBELL, H.: Studies on N-allylnormorphine in man: antagonism to morphine and heroin and effects of mixtures of N-allylnormorphine and morphine. Amer. J. med. Sci. 231 (1956), 1.

GRUBER JR, C. M. and GRUBER, C. M.: N-allylnormorphine as an antagonist to the intestinal spasm produced by the addicting analgesics. J. Pharmacol. exp. Ther. 109 (1953), 157.

GUNNARSON, E.: The bile ducts of cholecystectomised patients with and without dyskinesia before and after morphine injection. Acta radiol. 45 (1956), 298.

HART, E. R. and MCCAWLEY, E. L.: The pharmacology of N-allylnormorphine as compared with morphine. J. Pharmacol. exp. Ther. 82 (1944), 339.

HUGGINS, R. A., SPENCER, W. A., GEDDES, L. A. et coll.: Respiratory functions in man following intravenous administration of morphine N-allylnormorphine, and N-allylnormorphine after morphine. Arch. int. Pharmacodyn. 111 (1957), 275.

JACOBSSON, B., LANNER, L. O. and RÅDBERG, C.: The dynamic variability of the choledocho-pancreatico-duodenal junction. A roentgen cinematographic investigation. Acta radiol. 52 (1959), 269.

JONSON, M.: Width of common bile duct before and after morphine studied by cholegraphy. Acta radiol. 54 (1960), 345.

SCHAPIRO, H. and BEAL, J. M.: The effect of N-allylnormorphine on the choledochal sphincter action. Surgery 34 (1953), 870.

SMITH, J. L., WALTERS, R. L. and BEAL, J. M.: A study of choledochal sphincter action. Gastroenterology 20 (1952), 129.

UNNA, K.: Antagonistic effect of N-allylnormorphine upon morphine. J. Pharmacol. exp. Ther. 79 (1943), 27.

WINTER, C. A., ORAHOVATS, P. D., FLATAKER, L. et coll.: Studies on the pharmacology of N-allylnormorphine. J. Pharmacol. 111 (1954), 152.

WOODS, L. A.: The pharmacology of nalorphine (N-allylnormorphine). Pharmacol. Rev. 8 (1956), 175.

FROM ROENTGENDIAGNOSTIC DEPARTMENT D (DIRECTOR: DOCENT Å. LINDBOM), THE INSTITUTE OF RADIOPATHOLOGY (DIRECTOR: PROF. LARS SANTESSON), KAROLINSKA SJUKHUSET AND THE ROENTGEN DEPARTMENT (DIRECTOR: F. ULFSPARRE), OF KRONPRINSESSAN LOVISAS BARNSJUKHUS, STOCKHOLM, SWEDEN.

ANGIOGRAPHY OF ANEURYSMAL BONE CYST

by

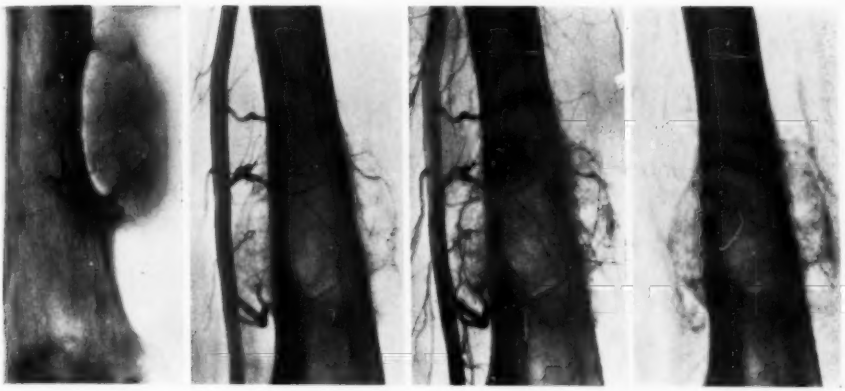
Å. LINDBOM, G. SÖDERBERG, H. J. SPJUT and O. SUNNQVIST

The roentgenographic appearances of an aneurysmal bone cyst have often been described in recent years and are generally regarded as characteristic (JAFFE, and SHERMAN & SOONG). A ballooned-out distention of the periosteum is usually outlined by a paper-thin subperiosteal shell of bone under which an eccentric destruction of both the cortex and the cancellous bone is evident.

The angiographic picture of this lesion has not been described in detail. SCHOBINGER et coll. reported arteriography of an aneurysmal bone cyst in a tibia. They found no changes in the arteries. During the venous phase, however, there was diffuse opacity of the cystic areas and the contrast medium was retained in the vessels of the involved area. The histologic appearances of the large distended or distorted thin-walled blood spaces in these lesions were described by JAFFE. He believed that the blood in these vascular spaces is not stagnant and that the large blood pool is being steadily drained and replenished. We have performed angiography of three aneurysmal bone cysts and these have shown uniform appearances. Some characteristic features have been observed.

The distal part of the femoral shaft of a 28-year-old man was involved in the first case (Fig. 1). In the second, the proximal metaphysis of a tibia of

Submitted for publication 25 July 1960.



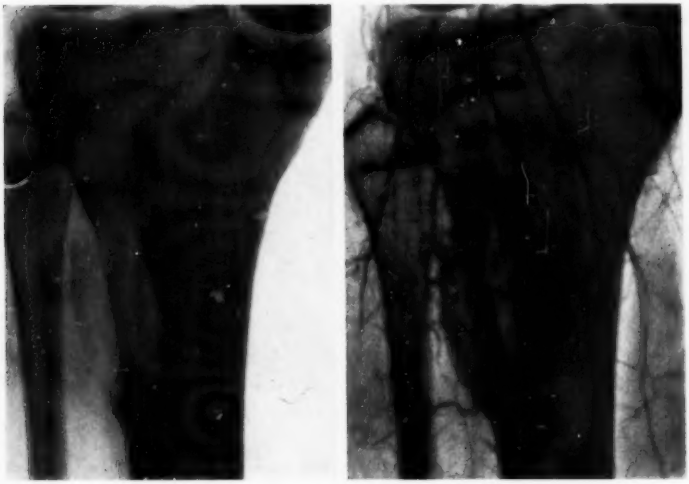
a b c d

Fig. 1. Aneurysmal bone cyst in distal part of femoral shaft of man aged 28. a) Cyst, surrounded by a thin shell of bone. b) Angiography, early arterial phase. Arteries leading to lesion contrast filled further peripherally than other arteries. c) Late arterial phase. Patchy opacity of cyst. d) Venous phase. Veins draining cyst contrast-filled.

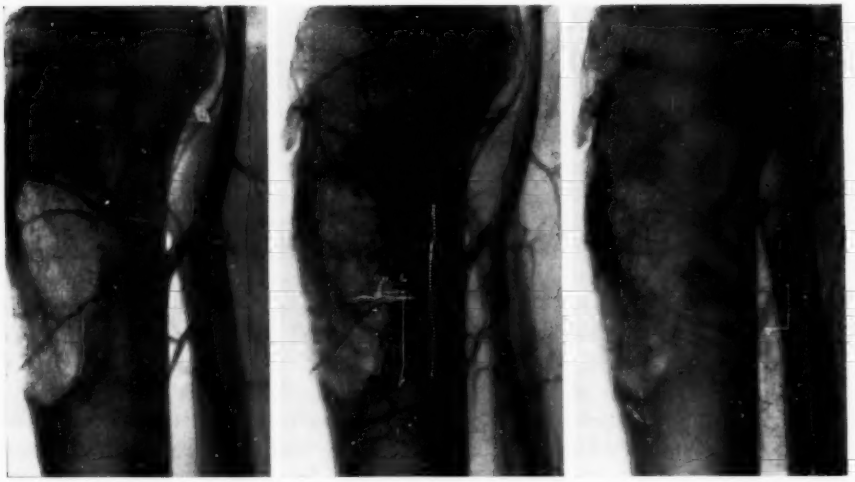
a 13-year-old girl was the site of the lesion (Fig. 2), and in the third case the proximal metaphysis of an ulna of a 16-year-old girl was affected. The femoral cyst was resected and the lesions in the ulna and tibia were curetted.

The histologic findings of the tissue from these three cases were typical. The loose fibrous tissue that formed part of the specimen was richly vascular and contained a large number of slightly dilated, thin-walled normal blood vessels. Several multinucleated giant cells and thin bands of osteoid were seen. Large and small cystic, often thin-walled, blood spaces varying from a few millimeters to several centimeters in diameter were characteristic features (Fig. 3). Giant cells were evident on the luminal aspect of the spaces, which they appeared to line. Narrow bands of osteoid often formed part of the wall of the blood spaces (Fig. 4). In addition osteoid and cancellous bone were sometimes found in the stroma with occasional spotty calcification peripherally. The cortical bone beneath the periosteum was attenuated; in some areas the stroma was purely fibrous or contained many giant cells along with osteoid and bone. Van Gieson staining of the sections failed to reveal the presence of elastic fibers in the walls of the small and large blood spaces of the lesions.

The angiographic examinations were all performed with a serial technique, in two of them an automatic changer being used. The arteries leading to the lesions were in two of the cases markedly dilated and in the other slightly dilated. During the passage of the contrast medium a slight but definite degree of opacity was noted throughout the whole area of the cyst and had



a b



c d e

Fig. 2. Aneurysmal bone cyst in proximal metaphysis of tibia of girl aged 13. a) Well delimited eccentric, superficial destruction and blown-out thin periosteal shell of bone. b) Arteriogram in late arterial phase. Identical projection as in (a). Opacity of whole cystic area. c), d), e) Serial arteriography, lateral. c) In early arterial phase, the arteries feeding the lesion are better filled than other arteries. d) In late arterial phase, patchy opacity of cyst. e) In early venous phase, the veins draining the cyst are already well filled, indicating an arteriovenous shunt.

Fig. 3. Low-power photomicrograph of resected aneurysmal bone cyst shown in fig. 1. × 6. Greatest part of lesional tissue consists of vascular spaces of varying size some of which contain blood residues. Periosteal shell of bone at upper border.

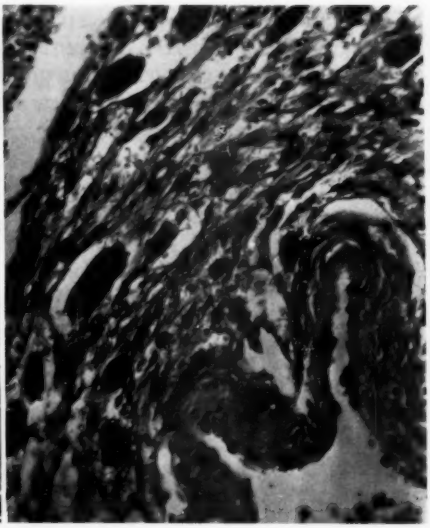


Fig. 4. Photomicrograph of curetted tissue from aneurysmal bone cyst. × 200. Loose fibrous tissue with many capillaries and some giant cells. Osteoid in wavy wall of collapsed large vascular space.

a certain patchy distribution (Figs 1 and 2). There was no peripheral hypervascularized zone as in malignant tumors. The veins leading from the lesion were filled somewhat earlier than the other veins indicating an arteriovenous shunt through the lesions; the degree of shunt was, however, considerably less than that in most malignant tumors. The patchy densities in the cystic area persisted late in the venous phase. The increased opacity must be due to the presence of contrast medium in the vascular lumina of the cyst. Whether the contrast medium passes through the many small vessels of the fibrous stroma or the large vascular lumina, or through both, cannot be gathered from the angiograms. We feel, however, that the patchy opacities could be explained by the presence of diluted contrast medium in the large vascular spaces.

The roentgenographic appearances of an aneurysmal bone cyst are in some cases atypical. When the thin blown-out shell of bone is absent, the roentgenographic changes may suggest a malignant tumor. Such tumors are, however, generally hypervascularized, especially peripherally. Thus in most instances it should be possible to differentiate a malignant tumor, e. g. fibrosarcoma or osteogenic sarcoma, from an aneurysmal bone cyst with the aid

of angiography. In the rare instance when the cyst is found in or adjacent to an epiphyseal region the differentiation from giant cell tumor in the ordinary roentgenogram is difficult. The angiographic appearances of giant cell tumor of bone as described by DOS SANTOS and others are characterized by a homogeneous but not patchy degree of opacity of the tumor area during the passage of the contrast medium through its vessels. The giant cell tumor contains an increased number of capillaries and these are too small to be observed directly in the angiogram. The presence of contrast medium in these vessels, however, causes the diffuse opacity of the tumor tissue. We made a comparison of the angiograms of 5 cases of giant cell tumor of bone with the angiograms of our three cases of aneurysmal bone cyst. In most of the giant cell tumors the opacity was somewhat more intense and more homogeneous than in the cysts. The angiographic appearances of these two different processes were, however, not obviously different in all cases. Angiography cannot therefore be relied upon for the differentiation between these two conditions.

SUMMARY

Angiography of an aneurysmal bone cyst may demonstrate a moderate hypervascularization of the lesion with patchy contrast filling of the cystic area and a moderate arteriovenous shunt. Three cases are described and discussed and the differential diagnosis is considered.

ZUSAMMENFASSUNG

Die Angiographie einer aneurysmatischen Knochencyste kann eine mässige Hypervaskularisierung der Erkrankung mit unregelmässiger Kontrastfüllung des cystischen Abschnittes und einem mässigen arteriovenösen Shunt zeigen. Drei Fälle werden beschrieben und diskutiert; die Differentialdiagnose wird besprochen.

RÉSUMÉ

L'angiographie d'un kyste anévrismal osseux peut montrer une hypervascularisation modérée de la lésion, avec opacification en taches de la région kystique et shunt artérioveineux modéré. Les auteurs en présentent et en étudient trois cas, et examinent le diagnostic différentiel.

REFERENCES

DOS SANTOS, R.: Arteriography in bone tumors. J. Bone Jt Surg. 32 B (1950), 17.

JAFFE, HENRY L.: Tumors and tumorous conditions of the bones and joints. Lea & Febiger, Philadelphia 1958.

SCHOBINGER, R. and STOLL, H. C.: The arteriographic picture of benign bone lesions containing giant cells. J. Bone Jt Surg. 39 A (1957), 953.

SHERMAN, R. S. and SOONG, K. Y.: Aneurysmal bone cyst. Its roentgen diagnosis. Radiology 68 (1957), 54.

FROM THE ROENTGENDIAGNOSTIC DEPARTMENT (DIRECTOR: PROF. OLLE OLSSON),
UNIVERSITY HOSPITAL OF LUND, SWEDEN.

RENAL OSTEONEPHROPATHY

by

STEN CRONQVIST

Renal osteonephropathy is a term used to describe skeletal changes sometimes seen in association with renal disease. The conditions of this disease may be fundamentally different. The most common and therefore most important condition is chronic renal insufficiency resulting from chronic glomerulonephritis, chronic pyelonephritis, or anomalies of the kidney or lower urinary tract. The second type consists of selective tubular disorders (DENT 1952).

The osseous changes are of various types. In the first cases published, represented by children, the osseous changes were regarded as rachitic and the term 'renal rickets' was used to define the condition (LUCAS 1883). Some of the cases were accompanied by dwarfism and sexual infantilism and were classified as renal dwarfism or renal infantilism (FLETSCHER 1911, BARBER 1920).

The roentgen findings in renal rickets were summarized by PARSONS (1927) and later by TEAL (1928), who distinguished two roentgenologic types: a rachitic and a 'woolly' type. In the former the changes were considered identical with those of rickets, in the latter they were thought to be characteristic of renal infantilism. In these cases the trabeculae of the bone were irregularly

Submitted for publication 13 June 1960.

thickened and poorly defined and the ossification was uneven which according to the authors gave the bone a stippled, woolly appearance. Alterations of this kind were observed in the advanced cases in which the bones were also deformed.

In one of TEAL's cases there was cortical erosion, which was regarded as constituting 'the most important feature in the radiographic appearance from a diagnostic point of view'.

NICHOLS & SHIFLETT (1934), in contrast to TEAL, claimed that the roentgen appearance of renal rachitis is not pathognomonic. Later reports began to appear of osseous changes in adults with chronic renal insufficiency, and these osseous alterations were found to coincide with those seen in osteitis fibrosa cystica. In advanced cases the roentgenographic appearance was identical with that of hyperparathyroidism, and the disease was called renal osteitis fibrosa generalisata or renal hyperparathyroidism (ALBRIGHT).

Skeletal changes. Two types of roentgen changes can thus be distinguished: one in which the lesions resemble those seen in rickets and one in which they resemble those of osteitis fibrosa cystica generalisata (SUSSMAN et coll. 1942). The former type occurs before, the latter mainly after, closure of the epiphyseal line. Occasionally both types may be found in the same patient. Ignorance of the possibility of this concurrence has been held responsible for the confusion in the interpretation of the roentgenographic appearance of renal osteodystrophy, particularly in children (DENT & HODSON 1954).

Before closure of the epiphyseal line the lesions are limited mainly to the distal part of the long bones, especially the radius and the ulna, where the metaphyses are increased in width, cupped, and of irregular outline (Fig. 10). In more advanced cases there is general rarefaction, obliteration of all compact tissue, and coarsening of the trabeculae in the cancellous bone. These changes may also occur in the skull, with granular rarefaction and splitting of the lamina interna and lamina externa, sometimes with thickening of the bone (Fig. 1). Deformation of the lower limbs may also occur, due either to 'softness' of the skeleton or possibly to epiphyseolysis, at times resulting in dwarfism (BRAILSFORD 1933).

A generalized or granular decalcification with or without disarrangement of the trabeculae may also be seen in adults, i. e. after closure of the epiphyseal line. Osteoporosis is described particularly in the older literature as a salient finding. Subperiosteal erosion is common and readily recognized. As mentioned, this erosion was considered by TEAL as an important diagnostic criterion of renal rickets, but CAMP & OCHSNER (1931) stressed the occurrence of the same type of change in hyperparathyroidism. Such erosion has since often been described in hyperparathyroidism and in renal osteodystrophy (PUGH 1951, GLASGOW et coll. 1955, DENT and HODSON 1954). The change is most frequently located in the middle phalanges of the fingers (Figs 2 and 5), but also occurs

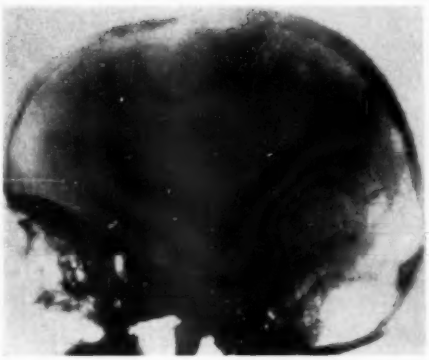


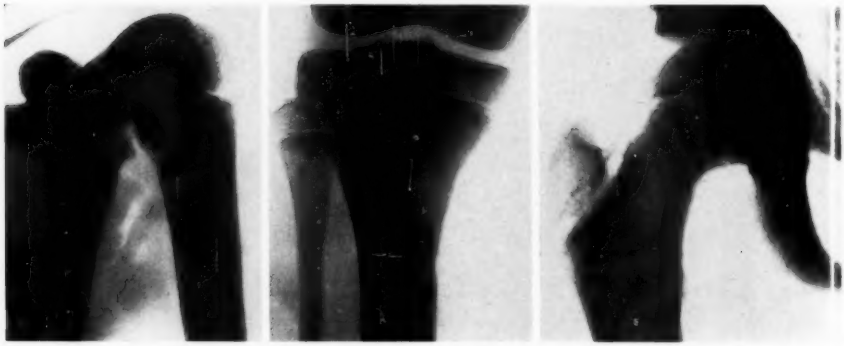
Fig. 1. Alternating sclerosis and rarefaction of skull. Calcification in the wall of the carotid siphon. Patient aged 34.

Fig. 2. Subperiosteal cortical erosion most marked in middle phalanges. Bone trabeculation altered. Small cysts in distal end of third metacarpal bone.

in the medial part of the upper third of the tibia, in the corresponding parts of the humerus and femur, in the acromion and lateral end of the clavicle and the distal part of the ulna (Fig. 3). Roentgenologically it is seen as a rarefaction, with a spicule-like arrangement of the cortex (Fig. 3c).

Resorption of the lamina dura (Fig. 4), and occasionally though rarely of the distal phalanges of the fingers, has also been described (Fig. 5). In advanced cases published in recent years the roentgen appearance was dominated by osteosclerosis (CRAWFORD et coll. 1954, BAKER 1954, BROOKFIELD et coll. 1955). The sclerosis may involve the major part of the skeleton, but it is most frequently localized to, and most easily recognized in the spine, particularly the lumbar spine. Sometimes a whole vertebra is sclerotic, while in other cases the sclerosis is present in broad zones adjacent to the superior and inferior surfaces of the body. Sometimes it is patchy, the alterations then contrasting sharply with the contiguous normal or even rarefied bone. The changes are often seen in the entire spine, but they may also be localized to a single vertebra (Fig. 6).

Metastatic calcification. Roentgen examination will sometimes also show calcification of soft tissue in various regions — metastatic calcifications (HUBBARD 1920, ANDERSEN et coll. 1942, MULLIGAN 1947, PENDERGRASS 1954). Three types have been distinguished: a 'physiologic', a 'vascular' and a 'pathologic'. In the physiologic type the calcium appears at those sites where acidotic excretion normally occurs, — such as lungs, stomach, kidneys. The vascular calcifications are present not only in the larger arteries of the body but may



a b c

d

Fig. 3.-Subperiosteal cortical erosion of humerus (a), of the upper medial part of the tibia (b), of the femur (c), and of the lateral end of the clavicle (d).

also be seen even in the small arteries of the extremities (Figs 5 and 7), which in more advanced cases appear as calcareous tubes. Calcifications situated around joints, in the synovia, and in or adjacent to tendinous attachments, are described (Fig. 8). According to some authors, such changes occur only in co-existing active arthritis. As a rule, metastatic calcifications occur in the patients with advanced osseous changes, but they may also be the first roentgen sign of renal osteodystrophy.

We have had the opportunity of examining a number of cases of chronic renal insufficiency showing osseous changes. The kidney diseases were: chronic pyelonephritis in 4 cases, chronic glomerulonephritis in 1 case and polycystic kidney disease in 1 case.

Fig. 4. Lower jaw. Absence of lamina dura.

Case reports

Case 1. Male, aged 17. Proteinuria and isosthenuria had been diagnosed at 8 years. Uraemia and severe leg pain at 15 years. The N. P. N. was then increased. The maximum specific gravity of the urine was 1 002. He had acidosis with a bicarbonate value of 13 m.mol. The serum calcium was somewhat below normal; the serum phosphorus, somewhat above. Urea clearance 9 %. The bicarbonate value returned towards normal on special treatment of the acidosis. At 17 years the N. P. N. was 200 mg %. He then had acidosis with a bicarbonate value of 13.8 m.mol. The serum calcium was 10 mg/100 ml, the serum phosphorus 7 mg/100 ml. The alkaline phosphatases rose from 29 to 91 units. After an infection, at 19 years, the patient became worse. N. P. N. 250 mg %. Acidosis with a bicarbonate value of 53.8 m.mol. The patient died.

Roentgen examination at the age of 17 had revealed small calcifications in both kidneys. The entire skeleton was abnormally dense and the trabeculae were thickened and coarse. The middle phalanges of the fingers and the medial proximal part of the humerus, femur and tibia showed marked subcortical rarefaction. The epiphyseal lines at the distal ends of the long bones were abnormally wide, and their outline was irregular and diffuse. Re-examination 2 years later showed a certain regression of the sclerosis in the metaphysis and the epiphyseal lines were no longer so wide or so diffuse (Fig. 10).

Autopsy revealed cystic kidneys with several pin-sized to hazelnut-sized cysts. Calcium deposits were seen in the tubular epithelium and in the cysts. Calcifications were also present in the pancreas, in sections from the intestine and in the spleen. The vertebral bodies and the sternum showed advanced osteoporosis. Between the trabeculae the connective tissue was loose and contained fibroblast-like cells.

Diagnosis: Polycystic kidney disease with uraemia.

The roentgen appearance was dominated by sclerosis. Wide-spread subperiosteal erosions were seen. Changes of the rachitic type responded well to special treatment of acidosis. In this case the bone pain was the outstanding symptom.

Case 2. Female, aged 36. At 8 years she had undergone operation with extirpation of the left part of a horseshoe kidney because of hydronephrosis. At 22 years hydronephrosis of the right part of that kidney and proteinuria were diagnosed. N. P. N. 35 mg/100 ml. Specific

gravity of urine 1 017. Proteinuria: max. 0.08 mg/100 ml. Five years later joint pain developed. The N. P. N. was then 70 mg/100 ml. Urea clearance 8 %. The following year the chronic renal insufficiency became worse. The N. P. N. was 195 mg/100 ml, serum calcium 4.5 m. E. q., serum phosphorus 10.4 mg/100 ml. Alkaline phosphatase 13 units. The lowest bicarbonate value noted was 12.2 m.mol. The patient died 36 years old.

Roentgen examination in 1955 had revealed widespread calcifications in the arteries of the abdomen and extremities. Signs of sclerosis were seen in the lumbar spine and subcortical erosions in phalanges of the hands and feet. Right kidney was diminished in size.

Autopsy. The right kidney showed marked interstitial fibrosis with calcifications. Wide-spread interstitial calcifications were also seen in the lungs. Sections from the finger phalanges showed a thin cortex with signs of active bone destruction with numerous osteoclasts and fibrous degeneration of the bone.

Diagnosis: Chronic pyelonephritis with uraemia.

Slight sclerosis and marked subperiosteal erosions were demonstrated, particularly in the middle phalanges of the fingers. Calcifications were found not only in the arterial walls but also in the kidneys and lungs. Joint pain was a prominent feature of the clinical picture.

Fig. 5. Resorption of the outer phalanges. Subperiosteal erosion of the middle phalanges with calcification of small vessels.

Case 3. Female, aged 54. At the age of 48 proteinuria and increased N. P. N. had been diagnosed. Specific gravity of urine 1 018. Urea clearance 21 % respectively 18 %. During the following years she was repeatedly admitted to hospital. In 1951, she was then 51 years, the serum calcium was 5.2 m. E. q. and the serum phosphorus 5.0 mg/100 ml. In 1952 the patient complained of joint pain. In 1954 the urea clearance was 7.4 %. Serum calcium 5.6 m. E. q. Serum phosphorus 6 mg/100 ml. Alkaline phosphatase 30 units. The N. P. N. gradually rose to about 100 mg/100 ml. She had acidosis during the major part of her illness despite continuous treatment. The patient died with cardiac insufficiency at the age of 54.

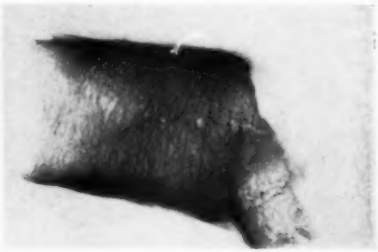
Roentgen examination. In 1947 the left kidney was small. Re-examination in 1952 revealed a ureteric stone and changes in the thoracic and lumbar spine with slight rarefaction and coarse trabeculae. In 1954 sclerosis along the superior and inferior surfaces of the vertebral bodies was noted. Similar changes were seen in other parts of the skeleton. Pathologic fractures were demonstrated in the right superior ramus and in the inferior ramus of the ischium. Erosions were seen on the medial upper aspect of the tibia.

Autopsy revealed small contracted kidneys with chronic pyelonephritis. The parathyroid glands were hypertrophic.

Diagnosis: Chronic pyelonephritis with uraemia.

Roentgenologically the lesions increased between 1952 and 1954. There was marked rarefaction and subcortical erosions were demonstrated. Acidosis had been present during the

a



b

Fig. 6

a) Lateral view of lumbar vertebral bodies showing sclerosis, especially adjacent to the intervertebral discs.

b) Post-mortem film of vertebral body showing same changes.

major part of the disease. Autopsy showed severe parathyroid hypertrophy despite absence of more advanced changes of the type of osteitis fibrosa generalisata. The patient had also had dull joint pain.

Case 4. Male, aged 25. At 10 years proteinuria had been diagnosed. At 19 years the N. P. N. was moderately increased. Specific gravity of urine: max. 1 014. Protein in urine: max. 0.5 mg/100 ml. At 24 years he had anaemia. The N. P. N. was then 100 mg/100 ml. He had a swelling in the left sternoclavicular joint and another in the left elbow. Biopsy specimens showed changes which were interpreted as metastatic deposition of calcium as a consequence of renal osteodystrophy. The following year the swelling increased and subcutaneous well-defined infiltrations occurred in different parts of the body surface. CO_2 = 50 vol %. Serum calcium 12.4 mg/100 ml. Serum phosphorus 9.0 mg/100 ml. Alkaline phosphatase: max. 16 units. N. P. N. max. 258 mg/100 ml. The patient died.

Roentgen examination. In 1953 large calcium masses adjacent to one of the sternoclavicular joints and in the soft tissues at the tip of the olecranon were demonstrated. Re-examination in 1954 revealed bilateral changes in the medial end of the clavicle. Fresh periosteal deposits were demonstrated on the left side. The structure in this region showed wide-spread destruction alternating with rarefaction. The trabeculae were partly eroded and partly sclerotic. Erosions were also seen in the ribs. The calotte showed rounded sclerotic areas. Skeletal changes with rarefaction alternating with sclerosis were generalized and observed in the entire lumbar spine, pelvic girdle, femur and lower leg. A fist-sized expanding process, fairly well defined and due to calcium deposits, was present in the right pubic region. Abundant calcifications were seen in the vessel walls. The kidneys were contracted.

Fig. 7. Man, aged 25. Excessive calcifications of vessels.

Autopsy showed a shrunken right kidney. The left kidney was small. Calcium deposits were seen in the tubular lumina in some parts of the kidney and in the basal membranes in others. All of the parathyroid glands were markedly enlarged. The glands were built up mainly of chief cells. There was wide-spread bony destruction in the form of lacunar resorption. The bone trabeculae in the marrow were narrow and eroded by lacunae. The bone marrow between the changed trabeculae were fibrous and sometimes the seat of numerous cells of osteoclastic type. The vessels showed calcifications.

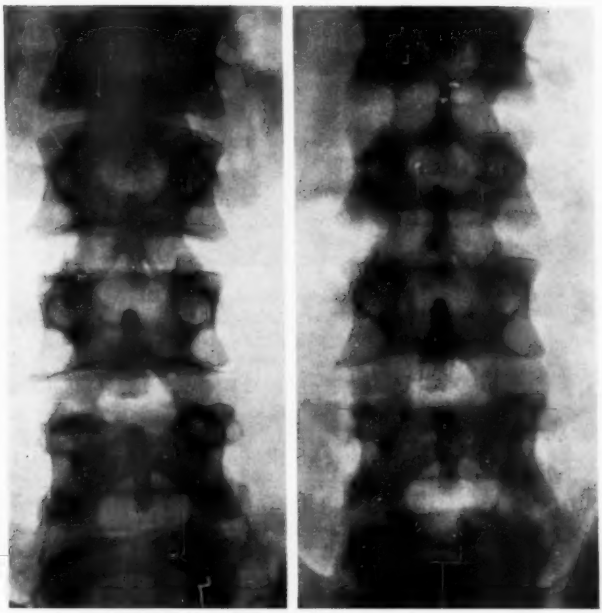
Diagnosis: Nephrosclerosis.

In this case the history was a long one and the picture dominated by metastatic calcifications. Marked skeletal changes were found with erosion and rarefaction alternating with sclerosis. Autopsy revealed severe enlargement of all the parathyroid glands, which were built up mainly of chief cells.

Case 5. Female, aged 37. Proteinuria and renal stone had been diagnosed at 22 years. The patient had had repeated attacks of pyelitis at 23. She had undergone operation for right renal stone at 24 years and nephrectomy at 25 for stone in the left kidney. Between the age of 34 and the age of 36 the N. P. N. varied between 60 and 80 mg/100 ml. During that period she complained of increasing stiffness of the legs and lumbar spine.

At 37 the N. P. N. was increased and she had isosthenuria. Specific gravity of urine 1 007. Urea clearance was 8 %. Acidosis with a bicarbonate value of 15.5 m.mol. The serum calcium was 5.6 m. E. q. Serum phosphorus 4.2 mg/100 ml. Alkaline phosphatase: 73 units.

Roentgen examination. In 1954 the patient, then 37, had wide-spread granular atrophy of the



a b

Fig. 8. a) Lumbar spine without definite osseous changes. b) Three years later. General sclerosis of vertebral bodies.

calotte. The lamina interna and lamina externa were poorly developed. The phalanges showed irregular rarefactions. Periosteal erosion was present in the middle phalanges of the fingers and a small cystic swelling in M3 of the left hand. Irregular rarefaction was also seen in the femur and humerus on both sides. The thoracic and lumbar spine as well as the pelvic girdle showed sclerosis.

Diagnosis: Chronic pyelonephritis with uraemia.

The patient had had proteinuria and renal stone at a fairly early age. She had generalized skeletal changes with a single cyst.

Case 6. Male, aged 29. At 17 years the patient had undergone left-sided nephrectomy for pyelonephritis with hydronephrosis. He had since had repeated attacks of pyelitis and during the following years proteinuria and renal insufficiency with falling clearance. N. P. N. about 100 mg/100 ml. At 29 years the serum calcium was 3.9 m. E. q. Serum phosphorus 6.0 mg/100 ml, bicarbonate value 16.1 m.mol. N. P. N. 110 mg/100 ml.

Roentgen examination. In 1955 when the patient was 24, small calcifications were demonstrated in the right kidney, which was abnormally small. Three years later he showed generalized sclerosis and subperiosteal erosions in the middle phalanges of the fingers. After a further 2 years the sclerosis and the subperiosteal erosions in the fingers had advanced.

Diagnosis: Chronic pyelonephritis with uraemia.

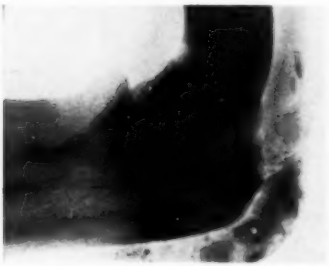


Fig. 9. Soft-tissue calcifications adjacent to elbow joint.

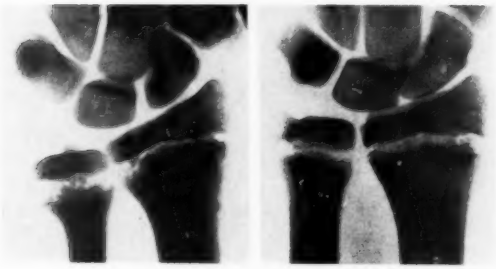
Case 7. Male, aged 48. At the age of 30 the patient had had chronic glomerulonephritis and in the following years proteinuria. In 1955, when 45, he had an acute exacerbation with anuria and the N. P. N. was then 150 mg/100 ml to 200 mg/100 ml. He was treated with the artificial kidney on repeated occasions and afterwards improved, and when he left hospital the N. P. N. was 60 mg/100 ml. He soon complained of fatigue and back ache as well as pain in the shoulders, hands, feet and knees. Serum calcium was 2.8 mg/100 ml. Serum phosphorus 7 mg/100 ml, bicarbonate 26.8. Four months later he felt worse. The N. P. N. was then 108 mg/100 ml. Serum calcium 1.9 m. E. q. Alkaline phosphatase was raised. The patient died.

Roentgen examination. In 1955 abnormally small kidneys but no skeletal changes were seen. In 1959 the kidneys had contracted still more and the entire spine was sclerotic.

Autopsy. Small contracted kidneys.

Diagnosis: Chronic glomerulonephritis.

It is generally recognized that chronic renal insufficiency is responsible for the osseous changes but the underlying mechanism is not properly understood. A base cannot, for example, be excreted in renal insufficiency, and the result will be acidosis and an increase in the concentration of the phosphate ions in the blood. Some authors believe the acidosis to be the direct cause of the skeletal changes (ALBRIGHT et coll. 1937). Others however consider that it is the relatively low calcium concentration in relation to the increased amounts of phosphate that prevents normal new bone formation (MITCHELL 1930). It is possible that an acquired vitamin D resistance may also be a contributory factor (STANBURY 1957). Changes in the ratio between the serum calcium and phosphorus have also been described as a cause of increased parathyroid activity with osteitis fibrosa generalisata as a consequence (BERGSTRAND 1921, GILMOUR 1947). Some authors believe that the primary cause of the syndrome, including the renal disease, is hypophyseal dysfunction (CHOWN 1935). It is probable that the skeletal changes are not caused by a single factor but result from the combined effects of two or more. An increased parathyroid activity thus may explain the subperiosteal erosion while the acidosis together with a change in the value of serum-Ca and serum-P may cause the rachitic-like changes. As mentioned, the type of skeletal changes in a given case generally depends



a b

Fig. 10. Chronic renal failure for 10 years in man, aged 18. Changes of rachitic type. a) Epiphyseal line widened and irregular, distal part of ulna and radius broadened. b) Regression on special treatment of the acidosis.

on the patient's age. Whether the severity or type of osseous changes vary with the degree of renal insufficiency is not known. It appears, however, as if even moderate renal insufficiency can with time cause skeletal changes. In all of the present cases the patients had a long history of renal disease. Raised alkaline phosphatase indicating skeletal destruction was noted in five. Acidosis was also noted in four cases. The roentgen findings in the author's cases agreed with those described in the literature summarized above.

Joint pain and/or stiffness were reported by most patients. These symptoms were the indication for roentgen examination in four cases. In three cases the pain was dull while in one it was stabbing.

In some cases the vertebral changes were detected incidentally in *routine films* of the kidneys or chest and prompted further investigation. In two cases the spinal changes were initially slight with generalized rarefaction and irregular coarse trabeculation. Roentgen control showed advancing sclerosis and the provisional diagnosis of renal osteonephropathy was confirmed (Fig. 9). Similar slight rarefaction has been seen in several cases of chronic renal insufficiency in elderly patients who could not be followed up roentgenologically. Senile osteoporosis could not be excluded in these cases. However, an incidental finding of even moderate osseous changes in patients with chronic renal disease should direct the examiner's thoughts to the possibility of renal osteodystrophy.

In the present material the youngest patient was 17 years old. A definite diagnosis of renal disease had been made 8 years previously and, as expected, both types of skeletal changes (rachitic and osteitis fibrosa cystica generalisata) were seen. A noteworthy finding in this case was the regression of the rachitic changes after protracted treatment of the acidosis (Fig. 10). Such regression has also been described by ALBRIGHT, for example, and has been taken as a

Fig. 11. Skeletal changes of the Paget type in a case of chronic renal failure.

sign that the acidosis is the main cause of the skeletal changes. Regression of the subperiosteal erosions after similar therapy has also been described, though no such improvement was observed in the present case.

Osteosclerosis of varying severity was seen in most of the cases. According to the pathologists a certain degree of sclerosis is regularly seen in patients with renal osteonephropathy — even in the absence of roentgenologically demonstrable sclerosis (GINZLER et coll. 1941, JAFFE 1942). This microscopic sclerosis is the result of a reactive new bone formation proceeding together with bone destruction; in the event of an increased rate of new bone formation, e. g. during remission, the sclerosis may be roentgenologically demonstrable. This is also the case in patients receiving intense calcipherol treatment (HOLMAN 1952).

Diagnosis. In the introduction it was mentioned that osseous changes may also occur in renal diseases not resulting in renal insufficiency. Such renal

diseases are Fanconi-Chiarii's disease and idiopathic hypercalcaemia, and others. They are all due to selective disorders of the tubules which are characterized biochemically by low to normal serum calcium, low phosphatase and normal or moderately raised phosphate values.

From a differential-diagnostic point of view, primary hyperparathyroidism offers the greatest difficulties. In this disease the osseous changes may be of the same nature as in renal osteonephropathy. Secondary chronic renal insufficiency due to deposition of calcium in the kidneys or stone formation is not uncommon. In primary hyperparathyroidism the serum calcium is raised and the serum phosphate depressed, while in renal osteonephropathy the converse changes are said to be most common though by no means the rule. In advanced cases of these two diseases the roentgenologic findings as well as the clinical picture may thus be identical, and then careful inquiry into the patient's history may help to establish the diagnosis.

Osteoporosis, common in elderly patients, is also an early roentgenologic sign of renal osteonephropathy. In elderly patients with chronic renal insufficiency, then, it is not always easy to decide the significance of the finding. The absence of subperiosteal erosions and normal calcium phosphate values are against renal osteonephropathy.

As known, the osseous changes in certain stages of hyperparathyroidism, renal osteonephropathy, and osteitis deformans (Paget) may be similar (LIGHTWOOD 1932, GUTMAN et coll. 1938, ANDERSEN et coll. 1942) (Fig. 11). The further course of the roentgen changes as well as the finding of normal serum-Ca and serum-P together with an excessive rise of the alkaline phosphatase in Paget's disease make a differential diagnosis possible.

The roentgen findings may be dominated by metastatic calcifications. But such calcifications are no more pathognomonic than the skeletal changes. Such changes are also seen in idiopathic hypercalcaemia, in Paget's disease, in hypervitaminosis D, and in this last mentioned condition bone changes resembling osteosclerosis may also occur.

In the final diagnosis of renal osteonephropathy in renal insufficiency, then, knowledge of the primary nature of the renal disease or of the malformation of the kidney is important. Low serum calcium and raised serum phosphate suggest renal osteonephropathy. None of the roentgen changes of renal osteonephropathy are pathognomonic but, like the metastatic calcification in cases of chronic renal insufficiency, osseous changes particularly of the rachitic type should guide the examiner's thoughts to renal osteonephropathy.

SUMMARY

Two types of skeletal changes in connection with chronic renal insufficiency, each of which appears to have a different pathogenesis, are described and discussed. An incidental finding of osseous changes in chest films or conventional films of the abdomen in cases of chronic renal insufficiency is suggestive of renal osteodystrophy.

ZUSAMMENFASSUNG

Zwei Typen von Skelettveränderungen in Verbindung mit chronischer renaler Insuffizienz, wobei jeder Typ eine verschiedene Pathogenese zu haben scheint, werden beschrieben und besprochen. Ein gelegentliches Vorhandensein von Knochenveränderungen auf Thorax- oder gewöhnlichen Abdomenröntgenbildern bei Fällen von chronischer renaler Insuffizienz ist auf renale Osteodystrophie verdächtig.

RÉSUMÉ

L'auteur décrit et analyse deux types de lésions squelettiques liées à l'insuffisance rénale chronique, chaque type de lésion semblant avoir une pathogénie différente. La constatation fortuite de lésions osseuses sur des radiographies du thorax ou de l'abdomen dans des cas d'insuffisance rénale chronique doit faire penser à une ostéodystrophie rénale.

REFERENCES

Albright, F., Drake, T. and Sulkowitch, H. W.: Renal osteitis fibrosa cystica. Report of a case with discussion of metabolic aspects. Bull. Johns Hopk. Hosp. 60 (1937), 377.

— Baird, P. C., Cope, O. and Bloomberg, E.: Study on the physiology of the parathyroid glands. IV. Amer. J. med. Sci. 187 (1934), 49.

Andersen, D. H. and Schlesinger, E. R.: Renal hyperparathyroidism with calcification of the arteries in infancy. Amer. J. Dis. Child 63 (1942), 102.

Baker, S. L.: General softening of bone due to metabolic causes. Brit. J. Radiol. 27 (1954), 609.

Barber, H.: Renal dwarfism. Quart. J. Med. 14 (1920—21), 205.

Bergstrand, H.: Parathyreoideastudien II. Acta med. Scand. 54 (1921), 539.

Brailsford, J. F.: Slipping of epiphysis of head of femur; its relation to renal rickets. Lancet 1933: I, p. 16.

Brookfield, R. W., Rubin, E. L. and Alexander, M. K.: Osteosclerosis in renal failure. Fac. Radiol. 7 (1955), 102.

Camp, J. D. and Ochsner, H. C.: Osseous changes in hyperparathyroidism associated with parathyroid tumor. Radiology 17 (1931), 63.

Cancelmo, J. J. and Bromer, R. S.: Osteonephropathy in children. Amer. J. Roentgenol. 65 (1951), 889.

Chown, B.: Renal rickets and dwarfism, a pituitary disease. Brit. J. Surg. 23 (1935—36), 552.

Crawford, R., Dent, C. D., Lucas, P. et coll.: Osteosclerosis associated with chronic renal failure. Lancet 1954: II, p. 981.

Dent, C. E.: Rickets and osteomalacia from renal tubular defects. Bone Jt Surg 34B (1952), 266.

— and Hodson, C. J.: Radiological changes associated with certain metabolic bone diseases. Brit. J. Radiol. 27 (1954), 605.

Fletscher, H. M.: Case of infantilism with polyuria and chronic renal disease. Proc. roy. Soc. Med. 4 (1911), 95.

Gilmour, J. R.: The parathyroid glands and skeleton in renal disease. Oxford University Press, London 1947.

Ginzler, A. M. and Jaffe, H. L.: Osseous findings in chronic renal insufficiency in adults. Amer. J. Path. 17 (1941), 293.

Glasgow, J. E. and Robinson, A. W.: Subperiosteal bone resorption in hyperparathyroidism. Brit. J. Radiol. 28 (1955), 381.

GUTMAN, A. B. and PARSONS, W. B.: Hyperparathyroidism simulating or associated with Paget's disease. Amer. Int. Med. 12 (1938), 13.

HOLMAN, C. B.: Roentgen manifestations of vitamin D intoxication. Radiology 59 (1952), 505.

HUBBARD, R. S. and WENWORTH, J. A.: A case of metastatic calcification associated with chronic nephritis and hyperplasia of the parathyroids. Proc. Soc. exp. Biol. 18 (1920—21), 307.

JAFFE, H. L.: Primary and secondary (renal) hyperparathyroidism. S. Clin. north. America 22 (1942), 621.

LEVIN, R. T. and GENOVESE, D. G.: Report of a case of longstanding renal insufficiency with extensive metastatic calcifications (renal osteitis fibrosa). Amer. J. Roentgenol. 64 (1950), 823.

LIGHTWOOD, R.: A case of dwarfism and calcinosis associated with widespread arterial degeneration. Arch. Dis. Childh. 7 (1932), 193.

LIU, S. H. and CHU, H. J.: Studies of calcium and phosphorus metabolism with special reference to pathogenesis and effects of dihydrotachysterol and iron. Medicine 22 (1943), 103.

LUCAS, R. C.: Form of late rickets associated with albuminuria, rickets of adolescence. Lancet 1883: I, p. 993.

MENVILLE, L. J., WILLIAMSON, L. and MATTINGLY, D.: Renal rickets with report of a case. Radiology 39 (1942), 410.

MITCHELL, A. G.: Nephrosclerosis in childhood with special reference to renal rickets. Amer. J. Dis. Child. 40 (1930), 101, 345.

MULLIGAN, R. M.: Metastatic calcification. Arch. Path. 43 (1947), 177.

NICHOLS, B. H. and SHIFLETT, E. L.: Renal rickets with report of case. Radiology 23 (1934), 677.

PARSONS, L. G.: The bone changes occurring in renal and coeliac infantilism and their relationship to rickets. Arch. Dis. Childh. 2 (1927), 1.

PENDERGRASS, E. P. and BROOKS, F. P.: Report of a case of osteonephropathy with vascular calcification in infancy. Radiology 62 (1954), 227.

PUGH, D. G.: Subperiosteal resorption of bone. Amer. J. Roentgenol. 66 (1951), 577.

STANBURY, S. W.: Azotaemic renal osteodystrophy. Brit. Med. Bull. 13 (1957), 57.

SUSSMAN, M. L. and POPPEL, M. H.: Renal osteitis. Amer. J. Roentgenol. 48 (1942), 726.

TEAL, C. G.: A radiological study of bone changes in renal infantilism. Brit. J. Radiol. 1 (1928), 49.

FROM THE ROENTGENDIAGNOSTIC DEPARTMENT (DIRECTOR: PROF. OLLE OLSSON),
UNIVERSITY HOSPITAL OF LUND, SWEDEN

NON-COLLAPSING AIR-FILLED ESOPHAGUS IN DISEASED AND POSTOPERATIVE CHESTS

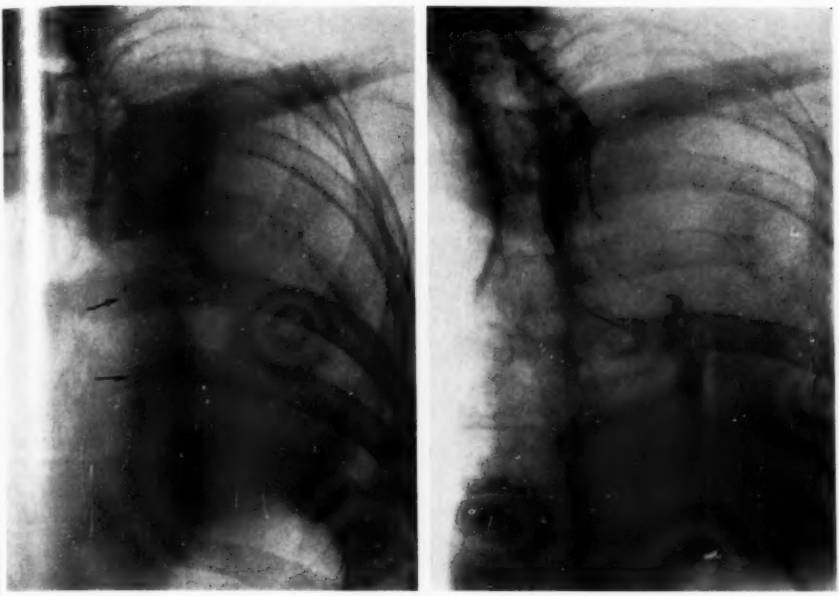
by

GUNNAR BLOMQUIST and PAUL S. MAHONEY

The esophagus is a self-collapsing organ which normally does not contain air. If, however, the walls of the esophagus become fixed by scarring and adhesions to surrounding structures, its lumen may remain patent and fail to collapse; the esophagus will then contain air and be demonstrable on roentgen examination of the chest. It is imperative that the esophagus be correctly identified and differentiated from other air-containing structures within the chest.

A review of the literature has revealed a few references to the roentgen appearances of the esophagus in inflammatory diseases and postoperatively. HAWES (1944) described in detail the roentgenographic changes in the esophagus in tuberculous mediastinitis. He mentioned one case in which the esophagus failed to collapse secondarily to inflammatory changes within the mediastinum. He did not illustrate this abnormality nor did he emphasize its significance in the differential diagnosis. KORNBLUM & OSMOND (1934) described the classic changes found in non-specific mediastinitis. They stressed the importance of an examination of the esophagus to complement the roentgen examination in all cases of mediastinitis. Deviation of a patent, rigid, barium-filled esophagus in the mediastinum was illustrated by DE LORIMIER, MOEHRING & HANNAN (1956) in a case of apical tuberculosis following thoracoplasty.

Submitted for publication 3 June 1960.



a b

Fig. 1. a) Air-filled deviated esophagus. Pleural margin of partially herniated right lung indicated by arrows. b) Barium examination of the esophagus.

These authors recognized that changes in the esophagus were the result of fixation of the esophageal walls to surrounding structures by scarring and adhesions, but they did not recognize that this structure often appears as a linear air column in an ordinary roentgenogram and that its nature can be confirmed by a barium swallow.

In recent years increasing progress has been made in thoracic surgery. The frequency of the occurrence of an air-filled esophagus secondary to mediastinitis has somewhat diminished as a result of early diagnosis and operation. Chest surgery, however, involves resection not only of pulmonary tissue but often mediastinal tissue as well. This fact, in turn, may result in non-inflammatory scarring and adhesions of the walls of the esophagus which prevent the lumen from collapsing. It is to this entity we wish to draw attention.

Ten cases will be described of an air-filled esophagus. These cases have been selected to illustrate the major roentgenographic features as well as to demonstrate the importance of the appearances in the differential diagnosis of chest conditions.

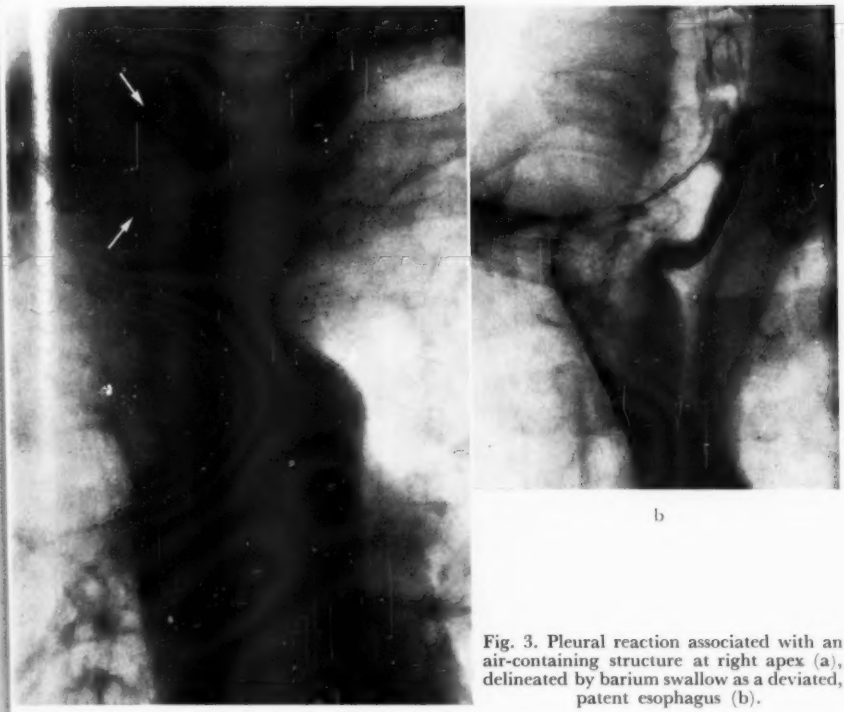
a b

Fig. 2. a) An air column partially overlaps the trachea. b) Barium examination of the esophagus.

Case reports

Case 1. Male, aged 49. A large tumor, surrounding and compressing the bronchus and displacing it towards the apical segment of the left lower lobe, was found at thoracotomy. Pneumonectomy was performed; the visceral and mediastinal pleurae were found to be adherent and both layers had to be removed; the aorta and the esophagus were exposed. Roentgen examination four months later disclosed a linear air column with a sharp kink at the level of the carina extending beyond the confines of the mediastinum (Fig. 1a). Examination of the esophagus with barium confirmed this structure to be the esophagus (Fig. 1b).

Case 2. Male, aged 42, complaining of wheezing on exhaling. Tomography, bronchography, and bronchoscopy disclosed stenosis of the left main stem bronchus. An unsuccessful attempt was made to resect the bronchus and affect an end-to-end anastomosis but, in so doing, a portion of the mediastinal pleura was stripped away. Left pneumonectomy was performed. Histology revealed chronic inflammatory tissue in the resected bronchus. Seven months later



a b

Fig. 3. Pleural reaction associated with an air-containing structure at right apex (a), delineated by barium swallow as a deviated, patent esophagus (b).

roentgen examination of the chest demonstrated a linear air column extending from the thoracic inlet to the left cupola of the diaphragm and partially superimposed upon the tracheal air column and the pleural margin of the herniated right lung (Fig. 2a). An examination of the esophagus with barium confirmed this structure to be the esophagus (Fig. 2b).

Case 3. Male, aged 62, underwent right middle and upper lobe lobectomy for adenocarcinoma. A large flap of the posterior mediastinal pleura was freed to cover the exposed hilum but the esophagus was exposed. Six weeks later roentgen examination disclosed right apical pleural reaction and an air-filled cavity in this region (Fig. 3a). A barium swallow examination showed that this structure was the esophagus (Fig. 3b).

Case 4. Male, aged 53, underwent left pneumonectomy for extensive bronchiectasis, this being followed postoperatively by a left hemothorax; this complication resolved spontaneously. One month later the esophagus was shown roentgenographically to be air-filled (Fig. 4a); this was confirmed by a barium swallow (Fig. 4b).

Case 5. Male, aged 56. Left pneumothorax induced for pulmonary tuberculosis of left upper lobe. Attacks of pleurisy then resulted in thickened pleura, most marked in the left

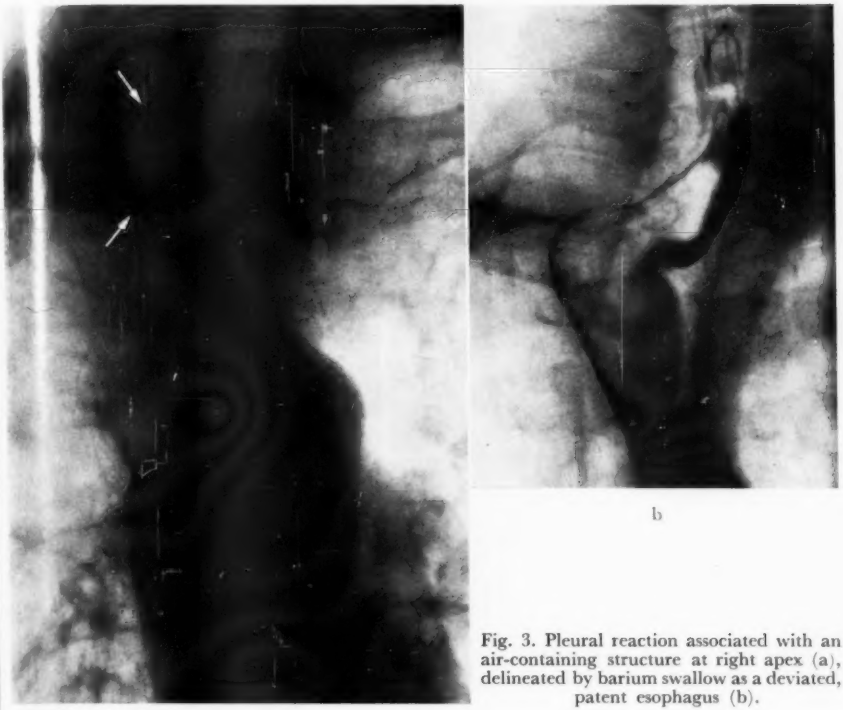
a b

Fig. 2. a) An air column partially overlaps the trachea. b) Barium examination of the esophagus.

Case reports

Case 1. Male, aged 49. A large tumor, surrounding and compressing the bronchus and displacing it towards the apical segment of the left lower lobe, was found at thoracotomy. Pneumonectomy was performed; the visceral and mediastinal pleurae were found to be adherent and both layers had to be removed; the aorta and the esophagus were exposed. Roentgen examination four months later disclosed a linear air column with a sharp kink at the level of the carina extending beyond the confines of the mediastinum (Fig. 1a). Examination of the esophagus with barium confirmed this structure to be the esophagus (Fig. 1b).

Case 2. Male, aged 42, complaining of wheezing on exhaling. Tomography, bronchography, and bronchoscopy disclosed stenosis of the left main stem bronchus. An unsuccessful attempt was made to resect the bronchus and affect an end-to-end anastomosis but, in so doing, a portion of the mediastinal pleura was stripped away. Left pneumonectomy was performed. Histology revealed chronic inflammatory tissue in the resected bronchus. Seven months later



a b

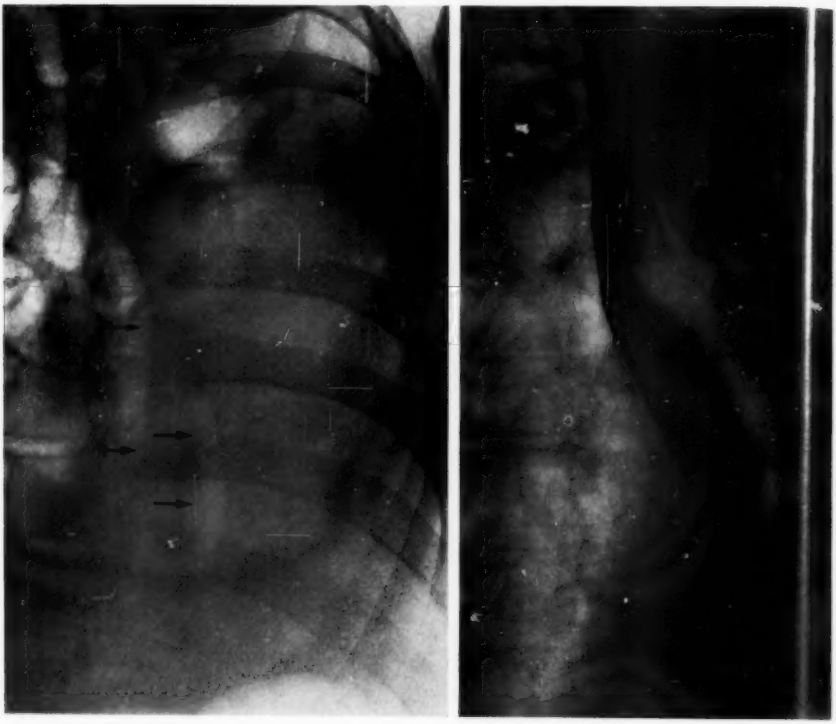
Fig. 3. Pleural reaction associated with an air-containing structure at right apex (a), delineated by barium swallow as a deviated, patent esophagus (b).

roentgen examination of the chest demonstrated a linear air column extending from the thoracic inlet to the left cupola of the diaphragm and partially superimposed upon the tracheal air column and the pleural margin of the herniated right lung (Fig. 2a). An examination of the esophagus with barium confirmed this structure to be the esophagus (Fig. 2b).

Case 3. Male, aged 62, underwent right middle and upper lobe lobectomy for adenocarcinoma. A large flap of the posterior mediastinal pleura was freed to cover the exposed hilum but the esophagus was exposed. Six weeks later roentgen examination disclosed right apical pleural reaction and an air-filled cavity in this region (Fig. 3a). A barium swallow examination showed that this structure was the esophagus (Fig. 3b).

Case 4. Male, aged 53, underwent left pneumonectomy for extensive bronchiectasis, this being followed postoperatively by a left hemothorax; this complication resolved spontaneously. One month later the esophagus was shown roentgenographically to be air-filled (Fig. 4a); this was confirmed by a barium swallow (Fig. 4b).

Case 5. Male, aged 56. Left pneumothorax induced for pulmonary tuberculosis of left upper lobe. Attacks of pleurisy then resulted in thickened pleura, most marked in the left

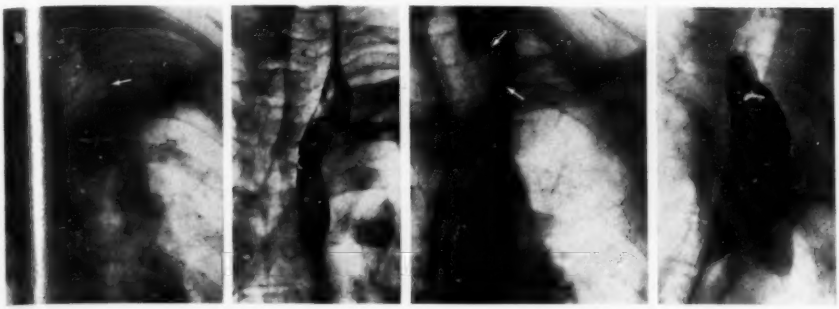


a b

Fig. 4. a) Air in esophagus (↑). Border of herniated right lung (↕). Fluid level at left apex due to hemothorax. b) A barium swallow shows the extent of the air-filling of the esophagus and differentiates it from the tracheal air column and the herniated right lung.

apical zone. Roentgen examination of the chest one year later revealed displacement of the mediastinum as well as a linear gas column lateral to the trachea (Fig. 5a); roentgen examination with barium showed this area to be part of the esophagus (Fig. 5b).

Case 6. Male, aged 42, developed pulmonary tuberculosis in the apical segment of the left lower lobe, which spread to the right lung. Recurrent attacks of pleurisy resulted in thickened pleura, most marked on the left side. Ten years later left lower lobe lobectomy and pulmonary decortication were performed. Two months later a roentgen examination of the chest revealed three air-containing structures at the left apex (Fig. 6a). The two smaller, round, thin-walled cavities overlapped one another and contained fluid levels; the third structure consisted of a linear air column extending along the left border of the trachea and contained no fluid level. A barium swallow showed the latter structure to be the esophagus (Fig. 6b). The two other structures were loculated pleural effusions.



a b

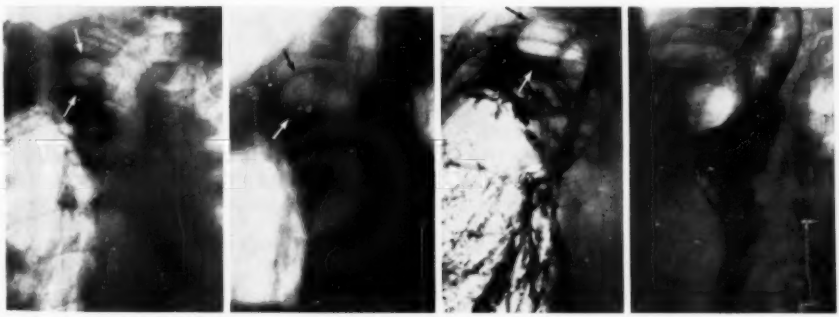
Fig. 5. Linear air column just lateral to the deviated trachea at left apex (a), confirmed by barium swallow as being the esophagus (b).

a b

Fig. 6. Two overlapping air-containing structures with fluid levels at left apex. Air-filled esophagus, lateral to the trachea, contains no fluid level. It is outlined with barium in (b).

Case 7. Male, aged 38, contracted pulmonary tuberculosis of the apical segment of the left upper lobe; thoracoplasty was performed. Roentgen examination of the chest one year later revealed a linear air space lateral to the trachea which could have represented herniation of the apex of the left lung. A barium swallow examination showed this structure to be the esophagus.

Case 8. Female, aged 60, developed pulmonary tuberculosis of the posterior apical segment of the right upper lobe (Fig. 7, a and b). The lesion regressed slightly over a period of a year under medical care. Bronchography was performed a year later. During the procedure some contrast medium flowed into an air-containing structure at the right apex. This was at first incorrectly interpreted as a residual tuberculous cavity filled via the bronchus to the posterior



a b c d

Fig. 7. a) Rounded air-filled structure just lateral to the deviated trachea at right apex. b) Tomography separates the structure from the trachea by a distance equal to only the thickness of the tracheal wall. Bronchography shows contrast medium outlining the structure. d) Postoperative barium swallow examination disclosed the air structure to be a portion of a dilated air-filled esophagus.

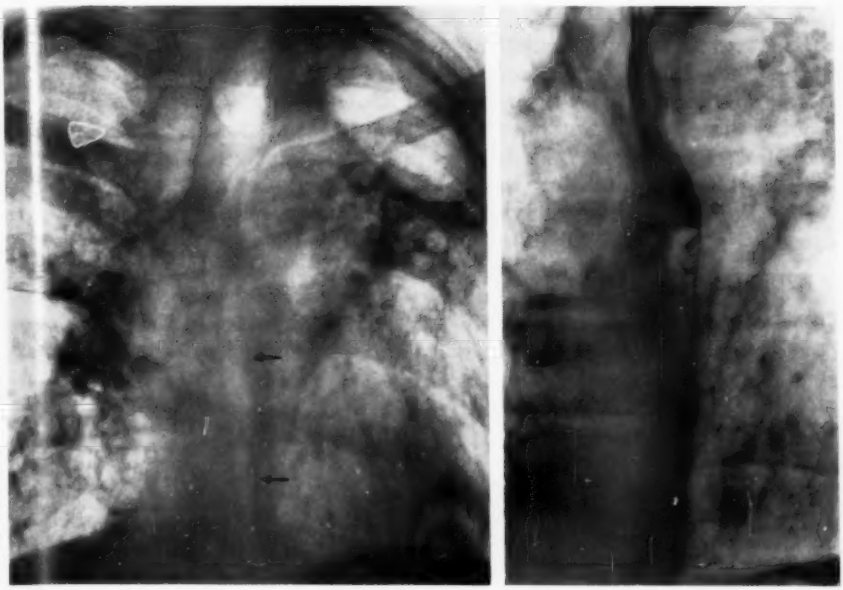
a b

Fig. 8. Linear air column to the right of trachea and extending obliquely across its lower end. b) The air-distended esophagus is well outlined in the right lateral decubitus position.

apical segment (Fig. 7c); in fact it was a portion of the esophagus. At subsequent operation when the right upper lobe was resected, it was noted that the serous and muscular layers of the esophagus had split and allowed the mucosa to herniate into the right apex. The exposed esophageal mucosa had become fixed by adhesions to the surrounding inflammatory tissue in the lung and failed to collapse upon itself (Fig. 7d).

Case 9. Male, aged 74. This case was diagnosed as being one of tuberculosis of the right upper lobe and was followed periodically. Nine years later roentgen examination disclosed slight displacement of the mediastinum as well as a linear air column just to the right of the trachea (Fig. 8a). A barium swallow examination in both the upright and lateral decubitus positions correctly identified this structure as being the esophagus (Fig. 8b).

Case 10. Male, aged 59, developed sarcoidosis and was treated with cortisone. Two years later the patient was found to have a left pulmonary abscess which was treated conservatively; the cortisone was discontinued and the abscess resolved slowly. Three years later roentgen examination disclosed a residual cavity at the left apex and marked fibrosis at the left hilum with retraction of the mediastinum to the same side; a linear air structure was interpreted as air in the esophagus (Fig. 9a). A barium swallow examination confirmed the nature of the structure (Fig. 9b).



a b

Fig. 9. Extensive fibrosis in the lung fields particularly on the left side with retraction of the mediastinum to that side. Air-filled esophagus outlined by arrows; abscess cavity at left apex. b) A barium swallow examination shows that the air column represents the esophagus. The lower part of the esophageal wall is thickened.

Discussion

An air-filled esophagus may sometimes be found in a roentgenographic examination of a diseased or postoperative chest. The inflammatory reaction about the esophagus may be confined to the mediastinum but more often involves the lung, pleura and mediastinum as well. The esophagus will appear as a vertical, linear air-containing structure about 1 cm in diameter, which is often curved, kinked and deviated to one side of the chest. Anatomically, the esophagus is well anchored in its upper third, but is more freely mobile in its lower two-thirds. The esophagus will consequently be found to be only slightly deviated in the upper part of the chest and often situated just lateral to the trachea; in the lower part of the chest, on the other hand, the esophagus is usually markedly deviated to one side.

Cases 1 and 2 are similar in that they both illustrate the typical appearance of the esophagus following an operative procedure within the thorax. In Case 1, the esophagus was most displaced below the carina in which situation

its diameter was also widest. This portion of the esophagus lay in the region of the mediastinum where the surgeon had removed the mediastinal pleura and exposed the esophagus to postoperative scarring and adhesions, allowing it to become mobile and deviate to the left. In Cases 2 and 3 the dissection exposed the esophagus and rendered it prone to scarring and retraction in the subsequent postoperative healing phase.

In Case 4, the esophagus was deviated for two reasons: the scarring following the operative procedure and the resultant forces secondary to cicatrix retraction initiated by a postoperative hemorrhage.

Cases 5, 6, and 7 illustrated the importance of a barium swallow examination to distinguish the esophagus from other air-containing structures. The air column in Case 5 could have been misinterpreted as herniation of the apex of the right lung. In Case 6, the two thin-walled air structures with fluid levels were by exclusion loculated pleural effusions. The third linear air column with no fluid level was, when filled with barium, shown to be the esophagus. The air column in Case 7 could readily have been mis-diagnosed as herniation of the apex of the left lung. A roentgen examination with barium confirmed it to be the esophagus.

Case 8 was a peculiar one in which an air-containing structure in the lung was incorrectly interpreted as a tuberculous cavity. The lesion actually represented a herniated part of the esophagus. During bronchography the patient inadvertently swallowed some of the contrast medium which subsequently filled the air structure and gave the impression that it was being filled via a bronchus.

Case 9 is included in the series to demonstrate the value of the lateral decubitus position to identify unknown air structures. In this case, the barium formed a pool in the structure and clearly identified it as the esophagus.

Case 10 demonstrates that two conditions occurring simultaneously within the thorax and both causing fibrosis and retraction, may complement one another in giving rise to air in the esophagus.

Anatomy and pathology. The anatomic position of the esophagus within the thorax renders the organ prone to involvement in any inflammatory reaction. Normally, the esophagus is a self-collapsing organ, flattened in its anteroposterior diameter and lying retrotracheally in front of and slightly to the left of the vertebral column. Its circumference is not of any great magnitude, but because the esophagus extends from the thoracic inlet to the diaphragm, it presents a rather large surface area to a great number of anatomic structures. The esophagus is consequently prone to involvement in any pathologic process arising in adjacent structures.

This concept is particularly apparent with regard to the mediastinal pleura. GLADNIKOFF (1948) has demonstrated roentgenographically that structures once described as 'mesenterium esophagei' are actually folds of mediastinal pleura

surrounding the esophagus. These folds may be seen as thin linear densities lying between the barium-filled esophagus and the air-containing lung. On the right side these pleural folds may be identified from the thoracic inlet to just above the right cupola of the diaphragm with a prominent bulge above the tracheal bifurcation corresponding to the arch of the azygos vein. On the left side the pleura may be visible from the thoracic inlet to below the aortic arch. These folds of pleura correspond to the anatomically described esophageal grooves in the right and left lungs respectively. Any inflammatory process which involves the mediastinal pleura will therefore subsequently involve the serosal surface of the esophagus as well.

Near the thoracic inlet the esophagus gives off numerous circular muscle fibers which are continuous with the inferior pharyngeal constrictor. Accessory slips of muscle fibers tend to fix the esophagus in position between the left mediastinal pleura and the pericardium. These supporting structures, which are more numerous and effective in the upper third of the esophagus, tend to anchor its position within the thorax.

Any process which tends to disrupt these supporting structures, such as surgery or a dissecting mediastinal inflammatory process, will free the esophagus and allow it to deviate to one side. Since these supporting structures are poorly developed in the lower part of the esophagus, the lower third will deviate more freely and to a greater extent than the upper third. Similarly, any inflammatory process involving structures in direct contact with the esophagus, such as the aortic arch, the left main stem bronchus, and the pericardium over the left atrium, will in turn directly affect the esophagus.

The walls of the esophagus may become rigid and fail to collapse in intrinsic conditions to which they are subject, quite apart from any extrinsic process in the mediastinal pleura. Scleroderma, in which the walls are infiltrated with fibrous tissue, may cause the esophagus to remain patent, and similar esophageal changes with the same result may occur in conditions like scirrhous carcinoma and achalasia of the cardia.

Differential diagnosis. The air-filled esophagus should be differentiated from air-containing structures such as loculated effusions, abscess cavities, lung herniations, and cysts. There are three roentgen features which are of importance in identifying the esophagus: (1) The often characteristic vertical, linear, and tortuous shape of the structure. The air column is usually of uniform diameter (approximately 1 cm) and is located along the course of the esophagus at all levels; (2) The absence of a fluid level; (3) The delineation of the esophagus with barium.

SUMMARY

An air-filled esophagus is often found on roentgen examination of diseased and postoperative chests and must be differentiated from other air-containing structures. Ten cases to illustrate this condition are presented and discussed.

ZUSAMMENFASSUNG

Ein luftgefüllter Ösophagus wird oft bei Röntgenuntersuchung von Thoraxerkrankungen und im Anschluss an Thoraxoperationen gefunden und muss von anderen luftenthaltenden Strukturen unterschieden werden. Zehn Fälle, die solche Zustände illustrieren, werden beschrieben.

RÉSUMÉ

Les radiographies de thorax pathologiques ou opérés montrent souvent un oesophage plein d'air, qu'il faut distinguer d'autres structures contenant de l'air. Les auteurs présentent dix cas illustrant cet état.

REFERENCES

DE LORIMIER, A. A., MOEHRING, H. G. and HANNAN, J. R.: Clinical roentgenology. Vol. 4. Charles C. Thomas, Springfield 1956.

GLADNIKOFF, H.: Roentgenographic study of the mediastinum in health and in primary pulmonary carcinoma. Acta radiol. (1948) Suppl. No. 73.

HAWES, L. E.: The roentgenological changes in the esophagus in tuberculous mediastinitis. Amer. J. Roentgenol. 51 (1944), 575.

KORNBLUM, K. and OSMOND, L. A.: Mediastinitis. Amer. J. Roentgenol. 32 (1934), 23.

FROM ROENTGENDIAGNOSTIC DEPARTMENT B (DIRECTOR: DOCENT N. P. G. EDLING) AND THE SURGICAL DEPARTMENT (DIRECTOR: PROF. J. ADAMS-RAY), KAROLINSKA SJUKHUSET, STOCKHOLM, SWEDEN.

CORRELATION OF ROENTGEN-ANATOMIC CHANGES AND FUNCTIONAL TESTS OF THE KIDNEYS IN HYPERPARATHYROIDISM

by

N. P. G. EDLING, C. A. EDVALL and C. G. HELANDER

In a previous paper (1960) the present authors reported on the relation of urography to inulin and PAH (para-aminohippuric acid) clearance and concentration tests performed in a series of cases of hyperparathyroidism. This paper reports a study on the relations of roentgen-anatomic changes in the kidneys, including size and the presence of calcifications, to the functional tests in the same material.

Material and Methods. The present series comprises 29 cases of hyperparathyroidism in which the blood calcium, initially elevated, reverted to normal after parathyroidectomy. Two of the patients were subjected to nephrectomy for concretions.

Routine roentgenograms of the kidneys were taken before and after the operation in 28 cases, and in one case only postoperatively. In many cases control examinations were performed before and after operation. As to the size of the kidneys in routine films, a length of 10 to 15 cm and a width of 5 to 8 cm are regarded as normal.

A total of 38 urographies in 18 of the patients are reported, all performed

Submitted for publication 16 May 1960.

with a contrast medium dose of 40 ml Urografin 60 % (sodium + methyl-glucamine diatrizoate). They were performed both before and after operation in 6 cases, and only postoperatively in 12 cases. The urographic density was estimated subjectively and termed either normal or poor. The degree of the contrast density of the pelves was the same in control examinations in the individual patients.

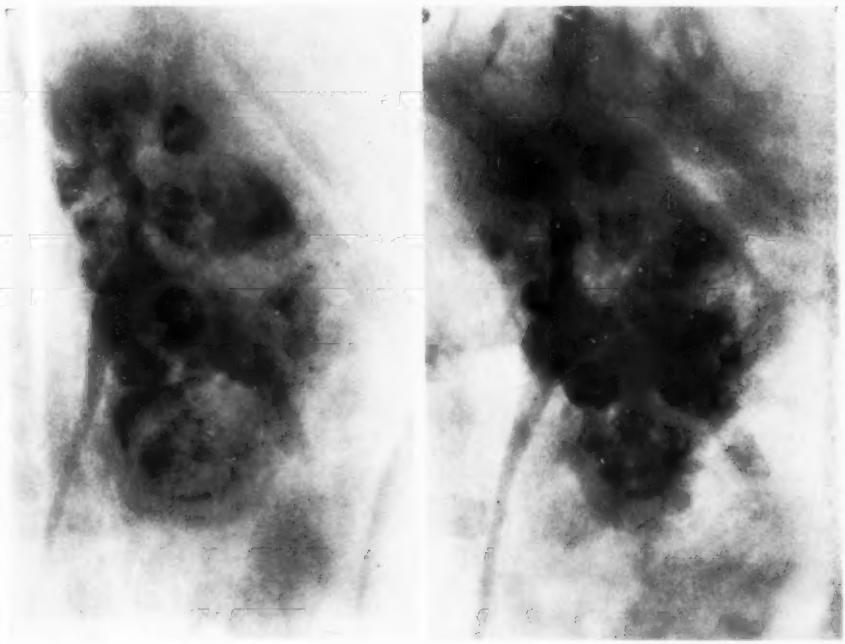
The analytic methods used for the clearance determinations have been described by EDVALL (1958). Bladder urine was used at all clearances except upon one occasion when selective examinations of the individual kidneys were done by ureteral catheterization. Normal values in both kidneys for inulin clearance are 92 to 146 ml/min, and for PAH clearance 400 to 700 ml/min. In the current series, inulin and PAH clearances were measured in 14 cases both before and after parathyroidectomy, in 5 cases only preoperatively, and in 10 cases only postoperatively; altogether 51 examinations were performed. In the cases examined both before and after operation, initially normal clearance values remained at normal limits and reduced values persisted postoperatively except in three cases. In these exceptions, either the inulin or PAH values were altered from normal to reduced or from reduced to normal postoperatively.

According to HELLSTRÖM (1959), the concentration power of the kidneys in hyperparathyroidism is lowered but improves in most instances after parathyroidectomy. Normal values are 1 025 and over. The concentration power of the kidneys in the present series was examined in 28 cases both before and after operation, and in one case only postoperatively; the concentration tests were performed several times in many of the cases. With the exception of one in which the value was at the lower limit of 1 025, the twenty-eight cases displayed reduced values preoperatively.

Results

The initial size of the kidneys in the present series before parathyroidectomy was as follows: larger than normal unilaterally in 2 cases (single kidneys), smaller than normal unilaterally in one case (pyelonephritic shrinkage), and normal bilaterally in the remaining 25 cases. When followed up during the course of the disease the size was found to be unchanged in 22 cases and decreased in both kidneys in 6 cases (Figs 1 and 2). The reduction in length was 1 to 2 cm and in width 0.5 to 1 cm. In the remaining case, examined after operation only, one kidney was non-excreting and of normal size while its fellow showed compensatory hypertrophy; both remained unchanged in size on follow-up examinations. Variations in size due to stasis are, of course, not included.

Renal calcifications were present in 25 of the 29 cases. Of these patients, 10 had bilateral nephrocalcinosis with or without lithiasis (Figs 1 and 2), and 15 had unilateral or bilateral multiple nephrolithiasis (the two single kidneys



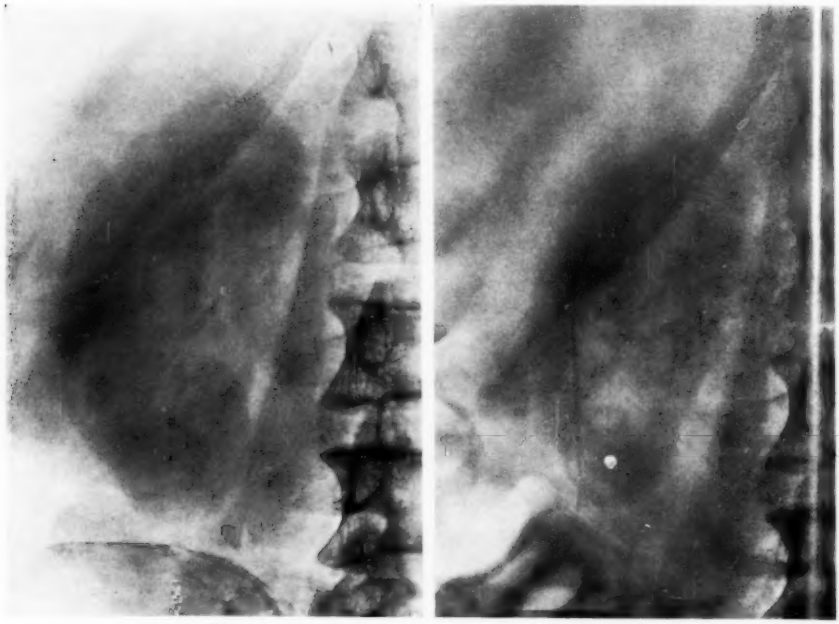
a b

Fig. 1. Hyperparathyroidism, left kidney. Parathyroidectomy in January 1957. a) In August 1953 the length of the kidney was 14 cm and the width 7.5 cm. Calcinosis. b) In May 1959 the length of the kidney was 12.5 cm and the width 6.5 cm. Reduction in the distribution of the calcifications.

included). The calcifications were passed in 3 cases during the course of the follow-up examinations but remained unaltered, or changed slightly in number and size, in the remaining 22 cases.

The six patients in whom kidney size decreased included 5 of the 10 cases of calcinosis, and one with a slight degree of unilateral lithiasis. The clearance values were lowered in all 6 cases. The concentration power, also low preoperatively, remained unchanged in 5, and improved without reaching normal limits in one case, postoperatively. The excretion on urography at 6 examinations performed in 3 of the cases showed density to be good in one and poor in 2 cases.

In the remaining 5 cases of calcinosis, the size of the kidneys did not change during the period of observation. The clearance values were normal in one and lowered in 4 cases. The concentration power was low in all cases preoperatively; it remained unchanged in one and improved in 4 — in one of



a b

Fig. 2. Hyperparathyroidism, right kidney. Parathyroidectomy in April 1954. a) In March 1954 the length of the kidney was 11 cm and the width just over 5 cm. b) In May 1957 the length was 9 cm and the width just over 4.5 cm. Slight calcinosis unchanged.

them to normal limits — postoperatively. Ten urographies performed in 4 of the cases showed density to be good in 3 and poor in one case.

In the 10 cases of calcinosis, then, the size of the kidneys decreased in 5, the clearance values were low in all but one, and the concentration power improved in 5 cases; urography showed density to be good in 4 and poor in 3 of the 7 cases so examined.

Of the 14 remaining patients with lithiasis, 5 had unilateral and 9 bilateral stones. The kidneys remained unchanged in size during the period of examination. The clearance values were normal in one of the cases with unilateral and in 5 of those with bilateral stones; in the remaining 8 cases the values were low. However, in 3 of the latter 8 cases, only one of the clearance tests gave reduced values either pre- or postoperatively. With the exception of one case with unilateral stones, the concentration power improved in all 14 cases examined both pre- and postoperatively, reverting to normal in more than half the cases.

I the 4 patients without calcifications, the kidney size remained unaltered. Th clearance values were normal in one and reduced in 3 cases. The concen ration power improved after parathyroidectomy in all 4 cases, reverting to r ormal in two of them.

T venty-four urographies performed in 9 of the patients with and 2 of those witl ut lithiasis showed density to be good in 7 of the former and one of the latte r, while it was poor in the 3 remaining cases.

Discussion

The kidneys are known to be affected adversely during the course of hyperparathyroidism. The presence of calcifications and the disturbance in the concentration power is discussed by HELLSTRÖM (1959), among others, and the disturbance in glomerular filtration and tubular excretion by EDVALL (1958). Closer study of the roentgen anatomy and function of the kidneys revealed further details.

In some of the cases there was a decrease in the size of the kidneys, suggesting shrinkage of the renal tissues. The process seemed to be slow, starting before the parathyroidectomy and continuing postoperatively. These cases had a higher incidence of calcinosis and of reduced renal function than the kidneys which kept their size. It follows that calcinosis of small kidneys in hyperparathyroidism is indicative of fairly severe renal damage.

EDVALL (1958) noted that the most advanced renal damage in hyperparathyroidism was found in patients with dysfibroplasia, whether or not they had calcifications in the kidneys. However, renal function was normal in only one of the cases of calcinosis in the present series, and in half of them the kidneys decreased in size. On the other hand, there was in the main no obvious difference as regards functional capacity between kidneys with and those without lithiasis.

Urography showed the density of the pelves to be good in some of the cases, despite the reduction in renal capacity. According to EDLING & coll. (1960) this is attributable to the damage to renal capacity in these cases not being more severe than can be compensated by technical measures such as dehydration of the patient and ureteral compression. Density was poor in a larger proportion of the cases of calcinosis than of those with or without lithiasis.

The incidence of kidneys with multiple calcifications in the series is very high. When present in a routine film renal calcifications therefore indicate testing of the blood calcium level.

SUMMARY

The relations between the size of the kidneys and the presence of calcifications on the one hand, and the inulin and PAH clearance, the concentration test, and urography on the other, were studied in 29 cases of hyperparathyroidism. A decrease in the size of the kidneys and the presence of calcinosis are usually indicative of reduced renal capacity.

ZUSAMMENFASSUNG

Die Beziehungen zwischen der Grösse der Nieren und dem Vorkommen von Verkalkungen einerseits, und dem Inulin- und PAH clearance, dem Konzentrationsversuch und der Urographie andererseits, wurden an 29 Fällen von Hyperparathyreoidismus studiert. Eine Abnahme der Nierengrösse und ein Vorhandensein von Kalzinose sind als Zeichen einer herabgesetzten Nierenfunktion anzusehen.

RÉSUMÉ

Les auteurs ont étudié dans 29 cas d'hyperparathyroidie les relations entre les dimensions des reins et la présence de calcifications d'une part, et les clearances à l'inuline et au PAH, l'épreuve de concentration et l'urographie d'autre part. La diminution des dimensions des reins et la présence de calcinose indiquent habituellement une diminution de la valeur fonctionelle des reins.

REFERENCES

EDLING, N. P. G., EDVALL, C. A. and HELANDER, C. G.: Correlation of urography and tests of renal function. Acta radiol. 54 (1960), 433.

EDVALL, C. A.: Renal function in hyperparathyroidism. Acta chir. scand. (1958) Suppl. No. 229.

HELLSTRÖM, J.: Primär hyperparathyroidism. (In Swedish.) Nordisk Med. 61 (1959), 551.

FROM THE ROENTGEN DEPARTMENT (DIRECTOR: O. SANDSTRÖM),
CENTRAL HOSPITAL, ESKILSTUNA, SWEDEN.

ROENTGENOLOGIC DETERMINATION OF THE LIVER VOLUME

by

L. WALK

Although the liver may be identified in the conventional roentgenogram, and methods to improve the visibility by means of pneumoperitoneum, contrast media, or inflation of the stomach or colon with gas, have been described, no adequate means of determining the size of the liver is to be found in the literature. Roentgenologic studies of the size (ref. 3 to 7, 10 to 14, 16, 19 to 21) or the volume (4, 10, 18) of the liver have included no autopsy controls, except a report of a liver index (20) which showed a considerable error of calculation. A method of measurement of the volume of the organ in experimental animals (1) is not adaptable for clinical purpose.

The size of the liver, estimated by clinical palpation, differs considerably from that found at autopsy (19); moreover the palpability varies from case to case and is difficult to evaluate (9, 15, 17). A roentgenologic method of determination of the liver volume, which is now described, provides an accurate means of mensuration and is a valuable clinical adjunct.

Material and Method

Roentgenograms of the liver were made immediately before opening the abdomen, and the roentgenologic measurements of the organ were compared to the postmortem specimen in a material of 80 autopsies.

Submitted for publication 11 July 1960.

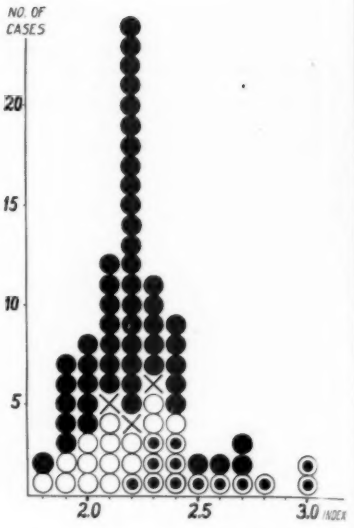


Fig. 1. Index for calculation of liver volume from linear dimensions of autopsy specimen. Analysis of 83 cases.

(●) Normal configuration of liver
(⊙) Thin borders
(○) Blunt thick borders
(×) Riedel's lobe

In order that the volume of the liver could be calculated from the linear size, the lateral (A), a. p. (B) and vertical diameters (C) of the liver specimen were recorded and the volume determined by immersion of the specimen in a basin of water and measurement of the overflow. The following formula was used:

$$\frac{A \times B \times C}{\text{Index}} = \text{Liver volume}$$

The diameters and the volume of the specimen being known, the index may be determined for any case (Fig. 1). The index, in a normal configuration of the liver, varies from 1.8 to 2.7 (average 2.25). Livers with a thin and flat left and/or right border have a greater index of 2.2 to 3.0 (average 2.6), whereas livers with blunt thick borders have a smaller index of 1.8 to 2.4 (average 2.1). Livers with a Riedel's lobe have a normal index.

The volume of the specimen can thus be calculated with a maximal error of $\pm$ 20 per cent if consideration is given to the form of the organ.

Roentgenologic mensuration

Because of the irregular oblique position of the liver in the abdomen, roentgenologic measurements cannot be directly compared to autopsy findings. A technique of measurement, minimizing the error caused by the oblique position of the organ, had to be found.

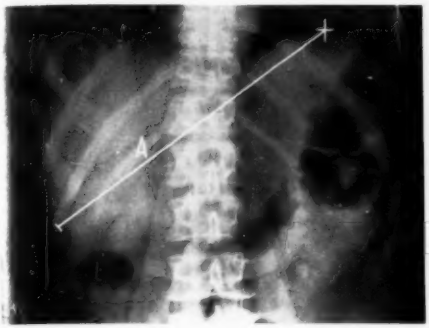


Fig. 2. Roentgenologic measurement of liver for calculation of volume, exposure I, diameter A.

Fig. 3. Exposure II, diameters B and C.

Fig. 3

The right border of the liver is easily identified in conventional roentgenograms. The lower surface is seen most distinctly where it abuts against the right kidney. The upper surface is not to be distinguished from the diaphragm, a fact which however causes only an insignificant error. The left border of the liver is difficult or impossible to localize; tomography does not much improve the visibility, and special methods of identification (2, 7) would make the measurement too complicated to be used in current work. The left border was therefore considered to lie in the plane of the middle of the left cupola of the diaphragm. As shown later, this is a satisfactory approximation and considerably simplifies the measurement.

Two roentgenograms are obtained at as great a focus-film distance as possible (150 cm in autopsy cases and 120 cm in clinical cases) to reduce the error of projection. Film size 30 × 40 cm.

Exposure I. The subject lies supine, with the tube vertically above the liver area and the film horizontal (Fig. 2). The distances of the focus to the film and to the middle of the patient's body are measured so that correction of the diameters in the roentgenograms to their actual size may be made.

Exposure II. The subject is rotated 50 to 60 degrees to the right, the film now being placed vertically (Fig. 3).

These positionings were employed in 51 autopsy cases but are also adequate for clinical use. The following further positionings have been tested in 29 autopsy cases: Exposure I^b, as I, but with the tube angled 20 degrees toward the head, and Exposure II^b, as II, but the tube angled 20 degrees toward the feet.

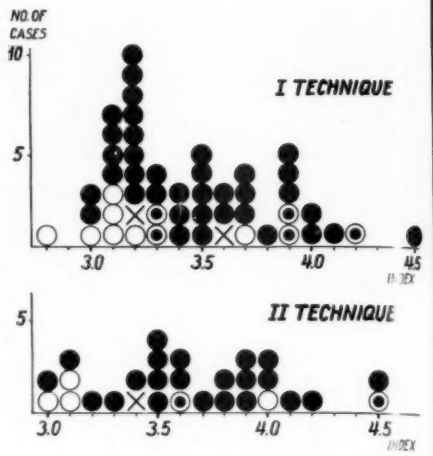


Fig. 4. Index for roentgenologic calculation of liver volume in an analysis of 51 autopsy cases examined by technique I and of 29 cases examined by technique II.

(●) Normal configuration of liver
(·) Thin right border
(○) Blunt rounded right border
(×) Riedel's lobe

Three diameters have been measured in the roentgenogram and corrected to their actual size: diameter A (exposure I), from the right border of the liver to the middle of the left cupola of the diaphragm; diameter B (exposure II), from the anterior border of the liver to the most distal part of its posterior surface as outlined by the diaphragm; diameter C (exposure II), from the lower surface of the liver, where it lies close to the upper pole of the kidney, in a transverse direction through the body of the liver to its upper surface at the lateral part of the diaphragm.

The liver volume has been calculated by the same formula as used for the autopsy material, but the index is different. Two techniques have been tried.

Technique I (exposures I and II, diameters A, B and C). The index has been determined in 51 autopsy cases (Fig. 4). The index, in a normal liver configuration, varies from 3.0 to 4.1 (average 3.55); with a thin flat right border of the liver, as seen in the roentgenogram (Fig. 5), it is 3.3 to 4.2 (average 3.75), whereas with a blunt thick border (Fig. 6) it is 2.8 to 3.7 (average 3.25). The index in one case was 4.5; the right lobe was pendulant but no characteristic thin border was seen in the film and the autopsy specimen revealed an anomaly of the left lobe which was narrow with an unusually small anteroposterior diameter.

These average sizes of the index, for any type of liver configuration, have been employed as follows:

1. Normal 3.55
2. Thin flat border ..... 3.75 (in marked cases 4.0)
3. Blunt thick border ..... 3.25.

Fig. 5. Thin right border of liver.

Fig. 6. Blunt rounded right border of liver.

The error of calculation in the great majority of our cases does not exceed ± 10 per cent. In a case of anomaly the error was 27 per cent, the actual volume being smaller than calculated.

Technique II (*exposures I^b and II^b, diameters A^b, B^b and C^b*). The index has been determined in 29 autopsy cases (Fig. 4) as 3.0 to 4.5 (average 3.75) for the normal liver, 3.6 to 4.5 (average 4.05) with a thin right border of the organ, and 3.0 to 4.0 (average 3.5) with a blunt thick border. The error has been ± 20 per cent, being less regularly distributed than in technique I.

The question has been considered as to whether the diameter A might be more exact if the left lobe of the liver could be identified in the roentgenogram. The position of the left border was recorded at autopsy and marked on the film (exposure I^b); the error of measurement was ± 17 per cent when compared with the autopsy diameter, whereas by measurement from the middle of the left cupola of the diaphragm it was ± 15 per cent. Identification of the left border of the liver would thus not improve the results; moreover, the method would become too complicated to be used in practice and would include an uncontrollable source of error.

The precision of the volume calculation can probably be increased by use of diameter A^b (exposure I^b), B^b (exposure II^b) and C (exposure II). In these three projections, any of the roentgenologic diameters has the smallest deviation from the autopsy diameter.

Application

The great variability of the liver on palpation in healthy adults has been pointed out in the literature (15); it may be palpable from 700 grams as weighed at autopsy although a non-palpable liver may be found to weigh up to 2 800 grams (19). In our material, the liver has been palpable and described as '1 to 3 fingerbreadths' from 1 200 ml displacement volume and '4 finger-

Table

Clinical palpation of the liver compared to the relative volume (volume of autopsy specimen of live per square meter of body surface) in an analysis of 50 autopsy cases

Liver on palpation	Relative liver volume				
	500—699 ml	700—899 ml	900—1 099 ml	1 100—1 499 ml	1 500 or more
Not palpable or palpable at costal margin	4	7	14	2	—
1 fingerbreadth	—	3	1	1	—
2 to 3 fingerbreadths	3	2	6	2	—
4 fingerbreadths or more	—	2	1	1	1

breadths' from 1 000 ml displacement volume. On the other hand, a non-palpable liver has been found to have a displacement volume of 2 500 ml at autopsy. This confirms that the evaluation of the liver by clinical palpation is difficult. The liver volume per square meter of the patient's body surface (relative volume of the liver) is more informative than the actual volume of the organ, and when compared to clinical palpation even the relative volume reveals the inadequacy of the palpation (see Table).

The relative volume has been determined in 304 cases by means of our technique I. It has varied from 520 to 1 850 ml.

Example of calculation. Diameter A is 31.5 cm, B 21.8 cm and C 13.2 cm in the film. The distance of the tube to the film is 120 cm and to the middle of the patient's body 102 cm. The corrected diameter A is 31.5: 120 × 102 = = 26.8 cm, B 18.5 cm, and C 11.2 cm. The liver has a rounded blunt right border, and the index 3.25 is chosen for calculation of the volume which is thus 26.8 × 18.5 × 11.2: 3.25 = 1 710 ml. The patient's height is 175 cm and weight 63.5 kg; from the diagram of Du Bois (Arch. Int. Med. 17 (1916), 863) the body surface is found to be 1.78 square meters. The relative volume is 1 710 : 1.78 = 960 ml (slightly increased).

The relative volume has varied from 550 to 860 ml in normal cases; a relative volume of 800 to 900 ml has been considered to be a borderline case, and when more than 900 ml to be enlarged.

In symptomless cholelithiasis the relative volume has been found to be normal, 575 to 850 ml, whereas in gallstones with colic and icterus it varied from 610 to 1 030 ml. In acute cholecystitis, it was as high as 960 ml; in 2 cases with a volume of 940 and 960 ml respectively, high diastase values indicated the presence of acute pancreatitis. In acute recurrent cholecystitis, the volume was up to 1 090 ml, greater than in ordinary cholecystitis; even here, a high

dia ase value was found in 2 of the 3 cases with the greatest volume. In cases pre iously operated on for biliary disease, and examined for cholangitis or for tones in the bile duct, the volume was from 615 to 1 010 ml. In diabetics, the volume was frequently increased, varying from 680 to 1 060 ml in pure cas , and 1 270 to 1 360 ml in 3 cases complicated by cardiosclerosis, cholan itis after cholecystectomy, and adipose degeneration of the liver (verified by iopsy), respectively. An increased volume was found in insulin-treated alcoholi s. The relative volume in hepatitis varied from 960 to 1 600 ml in the cou se of the disease. In cirrhosis, it varied from 550 to 1 550 ml. The relative volu me was up to 1 270 ml in myeloid leukemia, and up to 1 070 ml in lymphatic l ukemia; in polycythemia, there was no marked increase of the volume. In cardiac insufficiency, the relative volume was calculated as being from 810 to 1 180 ml. A marked increase in the volume is found in chronic pyelonephritis but normal values in chronic nephritis. A slight increase in volume is, for example, found in various other diseases and intoxications.

Follow-up examinations on 2 or 3 occasions were performed in 13 cases. The relative volume 2 weeks after cholecystectomy was 50 to 70 ml greater than before; the pre- and postoperative determinations in 3 cases were 720 and 770 ml, 880 and 935 ml, and 660 and 730 ml, respectively. The postoperative course in these cases was normal. In one case, with a probable subphrenic abscess following cholecystectomy, the relative volume after the operation was 185 ml greater than before (700 and 885 ml); the signs subsided after treatment with antibiotics. As the calculated liver volume will include subphrenic fluid if present, any marked increase in the volume after cholecystectomy may be of clinical significance. In one case in which cholecystectomy had been previously performed, the relative volume after removal of a stone from the bile duct was reduced by 110 ml (from 690 to 580). In a case of sprue in which a jejunocolic anastomosis had been performed because of terminal ileitis, the volume diminished from 1 400 to 1 180 ml during a year of medical treatment. Successive changes in the volume were also recorded in the remaining 7 follow-up cases, including a progressive increase from 810 to 860 and 890 ml in 18 months in a case of myelosclerosis.

SUMMARY

A method of roentgenologic determination of the liver volume, suitable for clinical use, is described. The error of the method is in the great majority of the cases less than ± 16 per cent. In occasional cases it may be 27 per cent, the actual volume being smaller than calculated.

ZUSAMMENFASSUNG

Eine Methode der röntgenologischen Volumbestimmung der Leber, für die klinische Arbeit geeignet, wird beschrieben. In der überwiegenden Mehrzahl der Fälle ist der Fehler der Methode höchstens ± 16 prozent. In einzelnen Fällen kann er 27 Prozent betragen, indem das Volumen kleiner ist als berechnet.

RÉSUMÉ

Description d'une méthode de détermination du volume du foie, convenant pour des explorations cliniques. L'erreur de la méthode est, dans la grande majorité des cas, inférieure à ± 16 pour cent. Exceptionellement elle peut atteindre 27 pour cent, le volume réel du foie étant inférieur au volume calculé.

REFERENCES

1. Adams-Ray, J., Hagberg, S., Hjelmström, P. and Olsson, S. E.: A roentgen stereophotogrammetric method for measurement of variations in the volume of organs in living animals. Angiology 5 (1954), 59.
2. Bétoulières, P., Carabalona, P., Pelissier, M. et Candon, J.: La pneumostratigraphie dans l'exploration du lobe gauche du foie. J. belge radiol. 39 (1956), 453.
3. Feldman, M.: Liver measurement. A comparative study on the relation of chronic gallbladder disease to the size of the liver. Radiology 10 (1928), 496.
4. Fiorioli, W.: Die klinische Auswertung des röntgenologischen Lebermessverfahrens. Med. Klin. 48 (1953), 1138.
5. Fleischner, F. G. and Sayegh, V.: Assessment of the size of the liver. New Engl. J. Med. 259 (1958), 271.
6. Köhler, A.: Totalröntgenogramme der Leber. Fortschr. Röntgenstr. 13 (1908), 145.
7. Lagarenne, P.: Délimitation et mensuration radiologique du foie et de la rate grâce à l'insufflation rectale du gros intestin. Thèse. Dijon 1920.
8. Lapeyre, N. C., Bétoulières, P., Carabalona, P. et coll.: L'étude radiologique du lobe gauche du foie. Intérêt de la pneumostratigraphie abdominale. J. Radiol. Electrol. 35 (1954), 478.
9. Lichtmann, S. S.: Diseases of the liver, gallbladder and bile ducts. Lea & Febiger, Philadelphia 1949.
10. Lutz, P.: Zur Technik der röntgenologischen Lebergrössenbestimmung. Med. Klin. 48 (1953), 1137.
11. Martin, C. L.: Roentgenologic studies of the liver and spleen. Amer. J. Roentgenol. 37 (1937), 633.
12. Mohr, W.: Röntgenologische Erfassung der Milz- und Lebergrösse bei Malaria. Fortschr. Röntgenstr. 72 (1949), 93.
13. Nimeh, W.: A new method for the determination of the size of the liver and spleen. Amer. J. Gastroent. 23 (1955), 147.
14. Osgood, C. W. and Habbe, J. E.: A clinical and roentgenologic study of factors influencing the palpability of the liver. Radiology 21 (1933), 66.
15. Palmer, E. D.: Palpability of the liver edge in healthy adults. U. S. armed Forces med. J. 9 (1958), 1685.
16. Pfahler, G. E.: The measurement of the liver by means of roentgen rays based upon a study of 502 subjects. Amer. J. Roentgenol. 16 (1926), 558.
17. Schiff, L.: Cited by Fleischner and Sayegh.
18. Teschendorf, W.: Lehrbuch der röntgenologischen Differentialdiagnose der Bauchorgane. Georg Thieme, Stuttgart 1954.
19. Zelman, S.: Liver and spleen visualization by a simple roentgen contrast method. Ann. Int. Med. 34 (1951), 466.
20. — Necropsy evaluation of the gas contrast roentgen visualization of liver and spleen. Amer. J. med. Sci. 236 (1958), 747.
21. — and Picard, C. M.: Roentgen and autopsy evaluation of percussion of the liver and spleen. Gastroenterology 29 (1955), 1037.

LABORATOIRE DE DOSIMÉTRIE DU SERVICE CENTRAL DE PROTECTION CONTRE LES RAYONNEMENTS IONISANTS (DIRECTEUR: PROF. L. BUGNARD), DE L'INSTITUT NATIONAL D'HYGIÈNE, PARIS, FRANCE.

CONTRIBUTION A LA MESURE DES RAYONS ROENTGEN DANS LE DOMAINE DE 5 A 50 KV

par

A. ALLISY et Mlle A. M. ROUX

L'installation générale comprend une source de tension stabilisée, un dispositif de mesure de la tension et du courant dans le tube à rayons roentgen ainsi qu'un électromètre destiné à la mesure des courants d'ionisation.

Générateur et appareils de mesure. Le tube à rayons roentgen, du type Machlett AEG 50 A, possède une fenêtre de béryllium d'environ 1 mm d'épaisseur et une anti-cathode de tungstène. Le courant maximal admissible dans ce tube est de 20 mA, la tension maximale de 50 kV. Le générateur haute tension analogue au modèle Massiot K 2 N, a été modifié de manière à ne fournir qu'une tension négative par rapport à la masse, l'anti-cathode du tube à rayons roentgen se trouvant obligatoirement à ce potentiel. Après modification, le générateur fonctionne en redresseur à une alternance. Nous y avons adjoint un condensateur d'une capacité de 0,1 μ Farad situé dans la cuve du voltmètre haute tension.

Une machine tournante suivie d'un stabilisateur magnétique à commande électronique, alimente l'ensemble de l'installation. Le chauffage du tube à rayons roentgen est pourvu d'une stabilisation supplémentaire formée d'un amplificateur qui voit à son entrée un signal proportionnel au courant anodique du tube à rayons roentgen et dont la sortie constitue une impédance variable disposée en série avec le filament du tube. Il est nécessaire de disposer à l'entrée de cet amplificateur un filtre passe-bas pour éviter des oscillations.

Soumis à la Rédaction 30 Mai 1960.

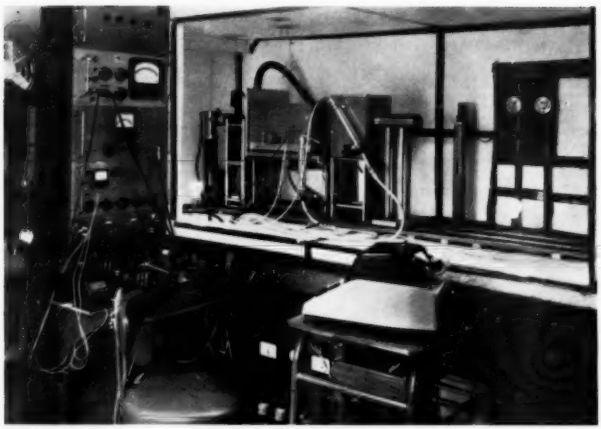


Fig. 1. Vue d'ensemble de l'installation de mesure des rayons roentgen très mous.

La haute tension est déterminée en mesurant le courant dans une résistance de 50 mégohms, ce courant atteint 1 milliampère sous une tension de 50 kV. La résistance globale est formée de 250 résistances de 200 kilo-ohms. Chacune de ces résistances est connue à 0,5 % près et peut dissiper une puissance de 2 watts. Le courant dans la chaîne de résistances ainsi que le courant dans le tube à rayons roentgen sont mesurés par une méthode d'opposition. La tension développée par ces courants aux bornes de résistances bobinées de haute précision est opposée à la force électromotrice d'un élément Weston. La sensibilité de cet appareillage de mesure a été déterminée pour contrôler la stabilité d'un point de fonctionnement plus que pour déterminer la valeur absolue des grandeurs à mesurer. Lors de la détermination des tensions, le galvanomètre dévie de 3 divisions pour une variation de 1‰ d'une tension de 5 kV et de 20 divisions pour une variation de 1‰ d'une tension de 50 kV. En mesurant le courant anodique du tube à rayons roentgen le galvanomètre dévie de 45 divisions pour une variation de 1‰ d'un courant de 5 mA. Les appareils de mesure qui viennent d'être décrits ainsi que la stabilisation du chauffage du tube à rayons roentgen sont disposés en dessous du banc de mesure (voir Fig. 1).

Mesure du courant d'ionisation. L'appareillage décrit est principalement utilisé à des fins de recherches sur les étalons, aussi les mesures sont-elles effectuées à l'aide de deux chambres d'ionisation: la chambre à étudier d'une part, une chambre de comparaison ou pilote d'autre part. Nous utilisons un système électrométrique décrit ailleurs (1) formé d'une lampe électromètre pentode et d'un galvanomètre monté en opposition dans l'anode de la lampe. Le système

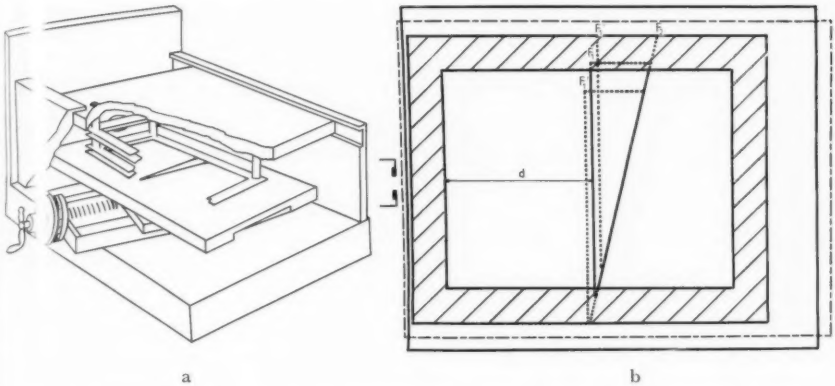


Fig. 2. Schéma perspectif (a) et, vue de dessus (b) de la chambre d'ionisation à parois d'air.

a été rendu symétrique en utilisant deux électromètres alimentés par la même source de haute tension hautement stabilisée. La source de tension variable ΔV destinée à la compensation de la charge des deux condensateurs de mesure, provient d'un potentiomètre de précision préalablement taré à l'aide d'un élément Weston. Cette tension ΔV est mise directement en série avec le condensateur chargé par le courant d'ionisation du pilote et par l'intermédiaire d'un diviseur de potentiel de précision en série avec le condensateur de mesure de la chambre étalon. Ce diviseur de potentiel qui travaille à impédance d'entrée constante, permet d'afficher tous les rapports, r, de 0 à 1 par bonds de 1/10 000. Le but de la mesure est alors de déterminer r de manière à compenser automatiquement la charge due au courant dans l'étalon lorsque l'on compense celle due au courant dans le pilote. La compensation de la charge due au pilote peut être manuelle ou électronique. Une reproductibilité de $\pm 0{,}5\,^0/_{00}$ est aisément obtenue avec ce dispositif. Les divers appareils de mesure ainsi que les alimentations stabilisées sont groupés sur un rack disposé à gauche sur Fig. 1.

Chambre d'ionisation pilote. Au cours de ce travail, nous avons trouvé que la chambre pilote la plus adéquate était formée par une deuxième chambre à parois d'air. La construction de cette chambre peut être beaucoup plus rudimentaire que celle de la chambre étalon mais il est important qu'il y ait l'épaisseur d'atténuateur minimale entre les plaques de mesure des deux chambres (6). Chaque fois que l'expérience le permet, le pilote est disposé derrière l'étalon (Fig. 1), le faisceau traversant les deux chambres est ainsi délimité par le même diaphragme. Cette géométrie peut difficilement être utilisée dans

la mesure des coefficients d'atténuation de l'air; dans ce cas, nous avons disposé une petite chambre pilote au voisinage de la fenêtre de béryllium du tube à rayons roentgen.

Lorsque le pilote est disposé derrière la chambre étalon, l'épaisseur d'air entre les deux plaques de mesure est de l'ordre de 30 cm. Cette couche d'air représente, à 10 kV, une atténuation de l'ordre de 60 %. La variation de cette atténuation est de 3‰ par degré centigrade, aussi avons nous été conduits à disposer le tube à rayons roentgen, le banc de mesure et les chambres d'ionisation dans une enceinte réalisée en polystyrène expansé de 5 cm d'épaisseur, à l'intérieur de laquelle la température est maintenue constante. Cette enceinte est visible sur Fig. 1.

I. Description de la chambre d'étude à parois d'air

Une vue perspective schématique de la chambre à plaques parallèles est donnée en Fig. 2a et une vue de dessus de cette chambre en Fig. 2b; on y a porté les positions extrêmes (représentées par des traits pointillés), et médiane (représentée en traits pleins), du système plaque de mesure-plaques de garde (Fig. 2b). L'ensemble plaques de garde-plaque de mesure forme un rectangle de dimensions: 30 cm × 25 cm. Les droites F_1, F'_1, F''_1 et F_2 passent par les milieux des fentes situées entre la plaque de mesure et les plaques de garde. F_1 et F_2 forment entre elles un angle d'environ 10 degrés, F_1 étant perpendiculaire au faisceau de rayons.

Le système plaque de mesure-plaques de garde peut subir une translation dans un plan horizontal parallèlement à la fente F_2 grâce à un dispositif porte-outil de tour qui est visible sur Fig. 2a. Les écarts de planéité de l'ensemble ne dépassent pas 1/100 millimètre. Le système de garde formé de cadres métalliques (zone hachurée Fig. 2b), est uniquement fixé à la plaque haute tension, l'ensemble pouvant être déplacé le long de l'axe du faisceau grâce à un système de glissières visibles sur Fig. 2a. La distance d (Fig. 2b) peut ainsi être modifiée. Les 6 cadres du système de garde ont des dimensions intérieures égales à 16 cm et 21 cm, leurs dimensions extérieures étant respectivement de 21 cm et 26 cm et leur épaisseur de 8/10 de mm; ils sont distant entre eux de 8,6 mm à l'exception des cadres du milieu distants de 13,6 mm.

L'espacement entre plaque de mesure et plaque haute tension est de 7 cm, la longueur des plaques de garde de 13,5 cm, la longueur moyenne de la plaque de mesure qui a la forme d'un trapèze rectangle est de l'ordre de 17 mm. Dans les deux positions extrêmes de mesure (F'_1 et F''_1, Fig. 2b), cette longueur est respectivement de 12 et 22 mm. Il est bien clair que la différence entre le courant d'ionisation correspondant à la longueur de 22 mm et celui correspondant à la longueur de 12 mm est équivalente à une mesure effectuée avec une plaque à bords parallèles ayant une longueur de $(10 - \varepsilon)$ mm, ε étant la largeur

de une des deux fentes supposées identiques. Dans le cas de notre dispositif, la largeur des fentes est éliminée lorsque l'on effectue une différence entre deux mesures, à condition que les fentes soient rigoureusement parallèles. Cette condition est facilement réalisable en pratique car les plaques de garde peuvent être fixées sur le plan de référence en utilisant la plaque de mesure comme cale. On réalise ainsi toutes les largeurs de fente désirables en déplaçant la cale plus ou moins le long d'un axe parallèle à F_2. La plaque de mesure est posée sur trois plots d'ambre. Un des plots vient placer son extrêmité conique dans un trou de la plaque, l'autre qui se termine en forme de couteau, s'engage dans une rainure et le troisième, hémisphérique, est tangent à la plaque de mesure. Ce support (point-trait-plan) permet de remettre toujours la plaque de mesure dans la même position. Il est commode, en particulier, pour déterminer l'influence du manque de planéité. En remontant la plaque de mesure de 14/100 de mm par rapport aux plaques de garde, nous avons mesuré une variation de 2 % du courant d'ionisation moyen (correspondant à une longueur de mesure de 17 mm).

Soit l, la longueur de la plaque de mesure au niveau du faisceau pour une position donnée de la graduation du porte-outil; cette longueur n'est, en général, pas très bien connue à cause d'un repérage par rapport à l'axe du faisceau qui est assez difficile. Par contre, la variation Δl, pour un tour de la poignée du porte-outil, peut être déterminée avec très grande précision à l'aide d'un comparateur. En relevant le courant d'ionisation, I, en fonction du nombre de tours, on obtient une droite dans la mesure où l'atténuation des photons primaires par l'air du volume de mesure est linéaire. Etant donné qu'il existe une relation linéaire entre le nombre de tours et la longueur l, l'équation de cette droite peut s'écrire:

$$I = pl + q \qquad (1)$$

En général, nous relevons expérimentalement 5 points espacés l'un de l'autre d'une valeur Δl de l'ordre de 2 mm. La connaissance de ces 5 points permet de trouver les constantes p et q à l'aide de la méthode des moindres carrés. Les écarts entre les points expérimentaux et la droite atteignent rarement 1,5‰. La quantité, $p = dI/dl$, qui représente l'ionisation par unité de longueur, permet la détermination de la dose d'exposition en roentgens.

La chambre d'ionisation qui vient d'être décrite a été conçue de manière à pouvoir être transformée facilement en chambre étalon. L'ensemble amovible formé par le chariot porte-outil, les plaques de garde et la plaque de mesure (voir Fig. 2a) peut être remplacé par un système de plaques de garde entourant une plaque de mesure de 15 mm de largeur à bords parallèles. La possibilité de déplacement de la plaque haute tension et du dispositif de garde est conservée, ce qui permet une réduction importante de la longueur d'air située entre le diaphragme et la plaque de mesure.

Tableau

Rayonnement	kV	mA	Filtration inhérent approximative	Filtration additionnelle mm Al	1ère CDA	2ème DCA	Facteur d'homogénéité	$a \times 10^3$	$b \times 0^6$
A	5	5	195 mm Air + 1 mm Be	—	91,5 mm Air	99,5 mm Air	0,92	—	—
B	10	5	195 mm Air + 1 mm Be	—	0,024 mm Al	0,031 mm Al	0,77	3,022	2,617
C	20	2	195 mm Air + 1 mm Be	—	0,051 mm Al	0,076 mm Al	0,67	1,578	0,1769
D	40	1	195 mm Air + 1 mm Be	—	0,066 mm Al	0,095 mm Al	0,70	1,158	0,114
E	30	2	195 mm Air + 1 mm Be	0,097	0,101 mm Al	0,141 mm Al	0,72	0,690	0,173
F	30	2	195 mm Air + 1 mm Be	0,202	0,144 mm Al	0,232 mm Al	0,62	0,519	0,0481
G	30	5	195 mm Air + 1 mm Be	0,502	0,349 mm Al	0,556 mm Al	0,63	0,258	— 0,0484
H	40	2	195 mm Air + 1 mm Be	0,599	0,550 mm Al	0,915 mm Al	0,60	0,1857	— 0,0364
I	40	2	195 mm Air + 1 mm Be	1,303	0,992 mm Al	1,328 mm Al	0,75	0,0972	— 0,00456

II. Conditions expérimentales

Le Tableau donné ci-dessus indique les diverses conditions expérimentales dans lesquelles les mesures ont été effectuées. Nous avons donné pour chaque rayonnement, caractérisé par une lettre, les conditions électriques, les filtrations inhérentes et additionnelles ainsi que les premières et deuxièmes CDA. En général, les CDA sont exprimées en mm d'aluminium, à l'exception du rayonnement émis sous 5 kV pour lequel les épaisseurs d'aluminium étaient trop faibles pour pouvoir être déterminées avec précision. Nous avons exprimé ces CDA en mm d'air (760 mm Hg, 20° C). Pour être plus explicite dans la caractérisation de la qualité d'un rayonnement, nous avons introduit le facteur d'homogénéité qui est le quotient de la première CDA par la seconde. Nous avons retrouvé (Fig. 3) les anomalies de ce facteur qui ont été mises en évidence et expliquées par Zieler (17, 18). En comparant nos valeurs de CDA en fonction de la filtration totale à celles de Zieler (18), nous avons remarqué qu'à filtration égale nos CDA étaient, en général, supérieures de l'ordre de 10 %. Cet écart peut s'expliquer par l'incertitude dans la détermination de l'équivalence béryllium-aluminium et air-aluminium ou par des différences de pureté de l'aluminium utilisé.

Une autre explication plausible de ces écarts peut être donnée par le fait que les composantes alternatives superposées à la haute tension semblent assez différentes dans les deux expériences.

Fig. 4 indique les valeurs de l'ondulation, r, en fonction de l'admittance de charge du générateur; r est défini (9) par:

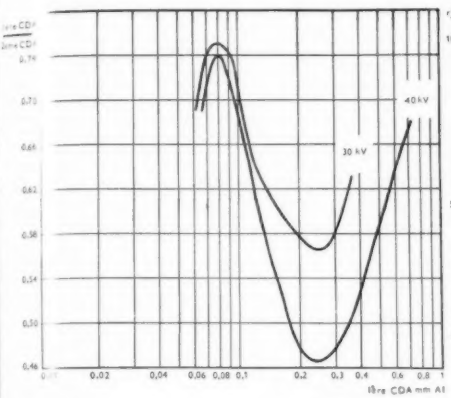


Fig. 3. Variation du facteur d'homogénéité en fonction de la CDA.

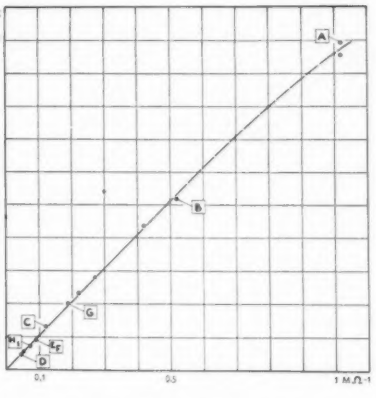


Fig. 4. Variation du ronflement en fonction de l'admittance de charge.

$$r\,\% = 200\,\frac{V_M - V_m}{V_M + V_m}$$

où V_M représente la valeur maximale, et V_m la valeur minimale de la tension du générateur.

Pour permettre la mesure de rayonnements très mous sans avoir les inconvénients d'un débit de dose d'exposition trop important et d'une géométrie incertaine, nous avons disposé entre la fenêtre de béryllium du tube à rayons roentgen et le diaphragme de la chambre étalon, un cylindre raccordé à une pompe à vide primaire (Fig. 1). Ce cylindre est fermé par deux fenêtres de téréphtalate de polyéthylène glycol (mylar), d'une épaisseur de 0,84 mg/cm² chacune. Ces fenêtres étant sur le trajet du rayonnement, il est intéressant de connaître l'équivalence air-téréphtalate en fonction de la qualité du rayonnement. Entre 5 kV, feu nu (rayonnement A-Tableau) et 40 kV, 1,3 mm Al (rayonnement I-Tableau), nous avons trouvé une équivalence constante à 3 % près. Nous admettrons dans ce domaine que 1 mg/cm² de téréphtalate est équivalent à 0,71 mg/cm² d'air.

III. Détermination des facteurs de correction de la chambre d'ionisation à parois d'air

A. Atténuation par l'air

Il existe un grand nombre de publications concernant l'atténuation de l'air (3, 4, 10, 12, 13, 18) mais les conditions expérimentales sont souvent difficilement comparables. Dans ce qui suit, nous donnerons l'atténuation de

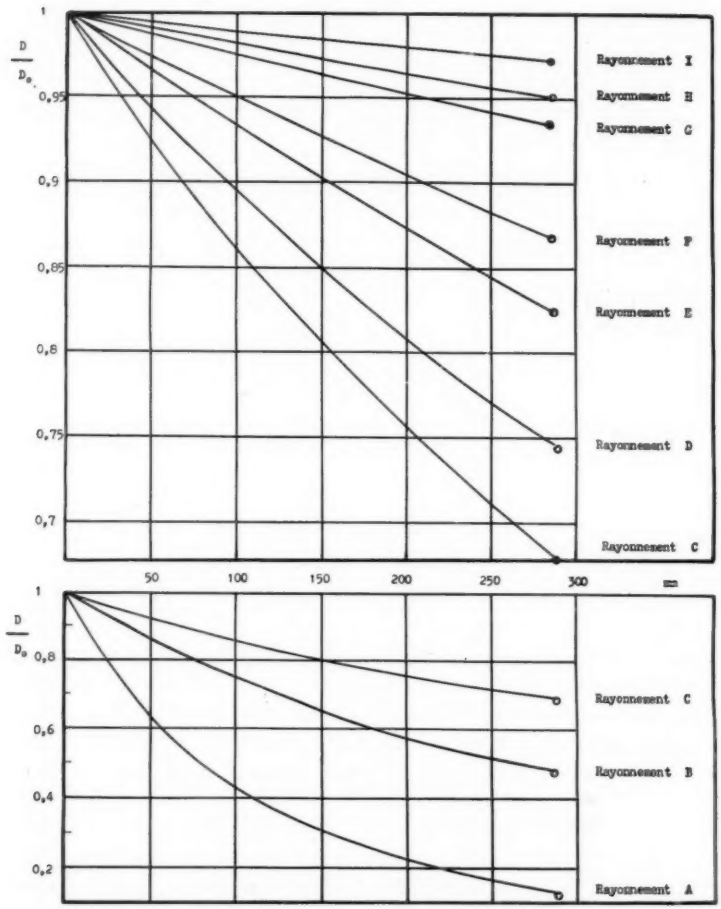


Fig. 5. Atténuation de la dose d'exposition par l'air à 20° C et 760 mm Hg. (Voir le Tableau, p. 62.)

la dose d'exposition par l'air. Les coefficients trouvés sont utilisables pour le calcul des corrections dans la chambre étalon; il ne faut pas les confondre, comme JAEGER (5) l'a montré, avec les coefficients d'atténuation de l'énergie déterminés, par exemple, à l'aide d'une chambre à absorption totale (2). Figs 5 et 6 donnent nos résultats. L'air qui a servi à la mesure était sec et les valeurs ont été normalisées à 20° C et 760 mm Hg. Les rayonnements utilisés sont caractérisés dans le Tableau, p. 62. Pour mettre en évidence l'absence

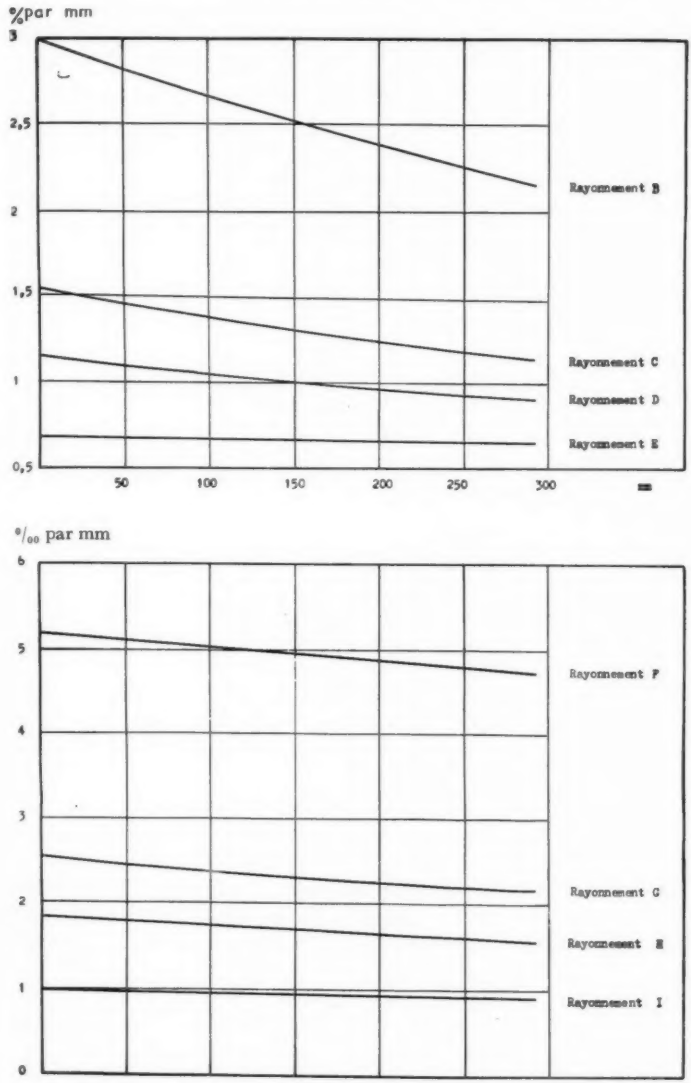


Fig. 6. Variation de l'atténuation de la dose d'exposition par l'air (20° C et 760 mm Hg) en fonction de l'épaisseur d'air traversé (l'épaisseur nulle correspondant aux conditions du Tableau, p. 62).

d'erreurs dues à la géométrie ou à la diffusion, nous avons mesuré l'atténuation dans 290 mm d'air par deux méthodes différentes. La première, utilisée pour le relevé des courbes (Figs 5 et 6), consistait à déplacer la chambre étalon en laissant le diaphragme fixe. Dans la seconde méthode, nous avons remplacé le vide dans le cylindre par de l'air à la pression atmosphérique (points expérimentaux, Fig. 5). Les deux méthodes ont donné des résultats concordants à 1‰ près. Etant donné la précision des résultats, nous avons recherché un moyen pratique et précis permettant de calculer les corrections dues à l'atténuation de l'air. Ceci est possible en remarquant que la fonction D_0/D (D = = dose d'exposition) varie sensiblement linéairement entre $l = 0$ correspondant à D_0 et $l = 300$ mm d'air sec à 20° C et 760 mm Hg. Par la méthode des moindres carrés, il est possible de mettre D_0/D sous la forme:

$$\frac{D_0}{D} = al + bl^2 + 1 \quad (l \text{ étant exprimé en mm})$$

Les coefficients a et b sont donnés dans le Tableau, p. 62. Le désaccord entre les points expérimentaux et la courbe ne dépasse jamais 1‰.

A partir de l'équation:

$$\frac{D_0}{D} = al + bl^2 + 1$$

il est facile de calculer la variation relative de l'atténuation de la dose d'exposition:

$$\frac{dD}{D \cdot dl} = \frac{a + 2\,bl}{1 + al + bl^2}$$

en fonction de l (Fig. 6).

B. Déformation du champ électrique

Les causes principales de déformation du champ électrique sont: le manque de planéité entre la plaque de mesure et les plaques de garde, la présence du système de garde et les différences de potentiel de contact entre plaque de mesure et plaques de garde. Les résultats concernant la première cause d'erreur ont été donnés plus haut. On se propose d'examiner dans ce paragraphe les deux autres causes d'erreur.

1. Déformation du champ électrique par le système de garde. Cet effet a été examiné pour diverses positions du système de garde par rapport à la plaque de mesure.

a) *Le système de garde est en position normale* (*Fig. 2b*). Pour effectuer cette étude, on a disposé, autour du système de garde, un blindage de dimensions: 31,4 cm × 23 cm (représenté par une succession de traits longs et de traits courts sur la figure), susceptible d'être porté alternativement au potentiel de

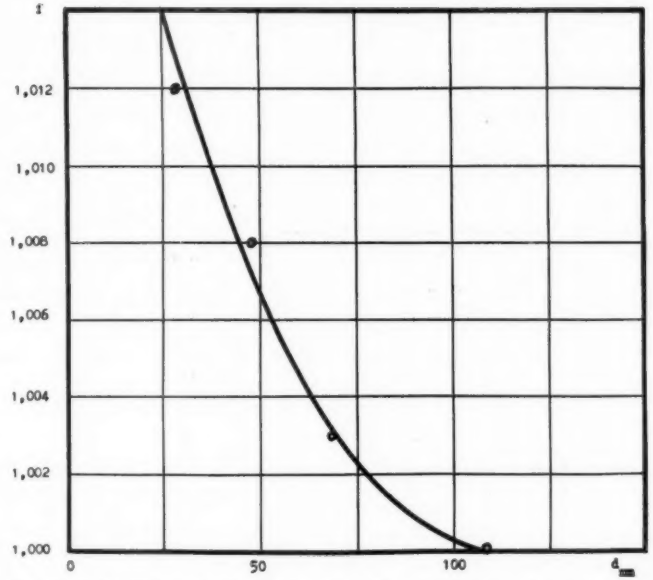


Fig. 7. Déformation du champ électrique. Facteur de correction en fonction de la distance entre système de garde et centre de la plaque de mesure (cf. la figure 2b).

la masse ou à celui de la haute tension. La construction de la chambre permettant de mettre en place ou de supprimer facilement le système de garde, on a d'abord déterminé, pour diverses longueurs de la plaque de mesure, les courants d'ionisation I_O et I_{HT} en l'absence de système de garde, le blindage étant relié successivement à la masse et à la haute tension. Ces mesures révèlent une augmentation de 7‰ des courants d'ionisation lorsque le potentiel du blindage passe de la masse à la haute tension. Les courants d'ionisation I_g sont ensuite déterminés avec le système de garde en place. On n'observe alors aucun écart lorsque le blindage est relié à la masse ou à la haute tension. Les valeurs de I_g se placent à mi-chemin entre I_{HT} et I_O à mieux que 0,5 ‰ près.

b) *Le système de garde est déplacé par rapport à la plaque de mesure.* Pour réduire l'atténuation de l'air, le système de garde peut se déplacer le long de l'axe du faisceau, la plaque de mesure et les plaques de garde restant fixes. Nous avons pu ainsi tester la déformation produite lorsque la distance d (Fig. 2b) varie. Les droites $I = pl + q$ ont été déterminées pour différentes positions du système de garde correspondant à des distances, d, variant de 108 à 28 mm. Les valeurs de I et de p diminuent lorsque la distance d diminue.

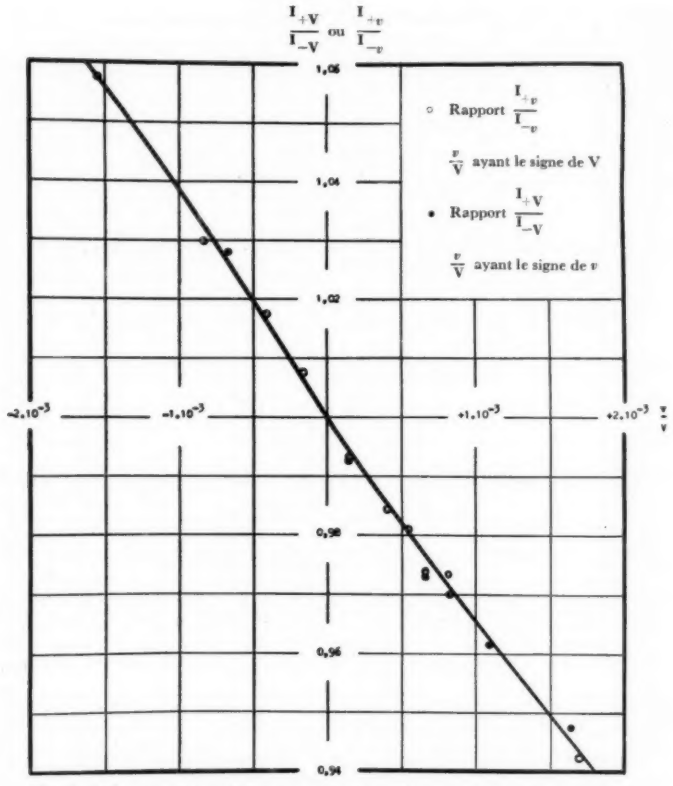


Fig. 8. Influence d'une différence de potentiel entre plaque de mesure et plaques de garde.

Nous avons tracé la courbe (Fig. 7) donnant le facteur de correction dans le cas d'une plaque parallèle de longueur $F'_1 F''_1$ (Fig. 2b) en fonction de la distance d. Ce facteur est égal à 1 lorsque le système de garde se trouve en position normale, c'est à dire pour $d = 108$ mm. La courbe a été déterminée à 20 kV (rayonnement C-Tableau p. 62).

Dans cette expérience, la distance entre l'extrêmité du cylindre vidé (portant le diaphragme) et la face avant du système de garde varie par suite du déplacement de ce système. Nous avons refait l'expérience en gardant cette fois la distance diaphragme-système de garde constante, ce qui nous a conduit à déplacer la chambre en sens inverse du système de garde d'une quantité égale. Les mesures alors obtenues ont été corrigées de l'atténuation de l'air sur la distance du déplacement de façon à pouvoir être comparées avec les mesures précédentes.

Le facteur de correction obtenu dans ce second cas, concorde avec celui obtenu dans la première expérience (Fig. 7) ce qui met en évidence l'influence négligeable du rayonnement diffusé pouvant provenir du diaphragme ou de la fenêtre de téréphtalate.

2. *Déformation du champ électrique par des différences de potentiel entre plaque de mesure et plaques de garde.* L'influence de ces différences de potentiel a été déterminée en portant la plaque de mesure (en position moyenne $l = 17$ mm), par rapport aux plaques de garde, à des potentiels, v, situés entre $\pm$ 150 mV et $\pm$ 1,5 volts.

Pour une valeur donnée, V, de la tension de collection, la plaque de mesure est portée successivement à un potentiel $+ v$ puis au potentiel $- v$ par rapport aux plaques de garde. On a mis en évidence le fait que le rapport:

$$\frac{I_{+v}}{I_{-v}}$$

des courants d'ionisation correspondants, est une fonction sensiblement linéaire de v/V (Fig. 8), à condition que V soit tel qu'il y ait saturation dans la chambre d'ionisation.

En imposant un potentiel v à la plaque de mesure, l'on a ensuite mesuré les courants d'ionisation I_{+V} et I_{-V}, correspondants à des valeurs de la haute tension égale à $+ V$ et $- V$. Après avoir corrigé les valeurs v d'une quantité due à une différence de potentiel de contact, les rapports $\frac{I_{+V}}{I_{-V}}$ se placent sur la droite déterminée plus haut (Fig. 8). Il est donc intéressant de déterminer l'erreur due à une éventuelle différence de potentiel de contact en mesurant les courants d'ionisation correspondant à des potentiels de collection $+ V$ et $- V$, les plus faibles possible, compatibles avec la saturation.

C. Influence des photons diffusés

La détermination exacte d'une dose d'exposition en roentgens, requiert la connaissance de l'ionisation due aux électrons produits par les photons diffusés hors du faisceau primaire; ionisation qui doit être déduite de celle mesurée dans la chambre.

D'après une technique mise au point par WYCKOFF (15), nous avons effectué les mesures de la contribution des photons diffusés à l'aide de tuyaux de téréphtalate de polyéthylène glycol de 12 mm de diamètre, d'épaisseurs différentes, alignés coaxiallement avec le faisceau de diamètre 4,5 mm.

L'épaisseur des parois de ces tuyaux doit être supérieure au parcours, dans le téréphtalate, des électrons primaires. Un de ces tuyaux est rendu conducteur par une application d'une mince couche de graphite colloïdal, il possède alors une épaisseur de 6,9 μ et peut être porté au potentiel milieu du système de

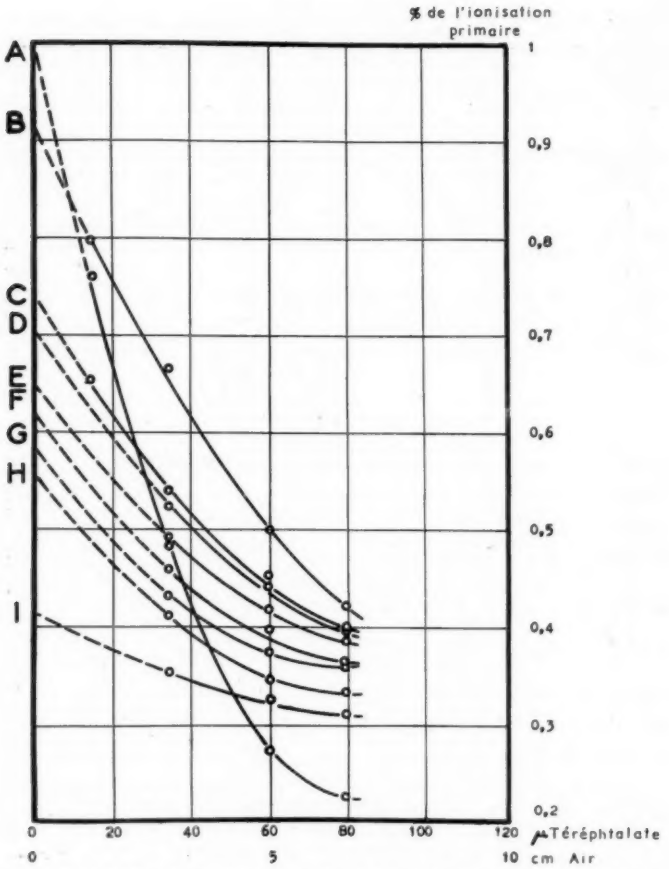


Fig. 9. Variation de l'ionisation relative due aux photons diffusés, en fonction de l'épaisseur des tuyaux de téréphtalate.

garde. On peut y insérer d'autres tuyaux de téréphtalate non graphités, d'épaisseurs croissantes (20 μ, 45 μ et 65 μ), ce qui permet de déterminer l'ionisation due à la diffusion en fonction de l'épaisseur. Nous avons auparavant déterminé la valeur de la distorsion du champ électrique causée par le tuyau de téréphtalate. Pour cela nous avons irradié entièrement la chambre à l'aide d'une source de radium et mesuré l'ionisation produite avec et sans le tuyau graphité (10). La différence entre les deux mesures se trouve être de 4,5 %.

Pour chaque rayonnement, nous avons établi la courbe donnant le quotient du courant d'ionisation, le tuyau étant en place, par le courant sans tuyau en

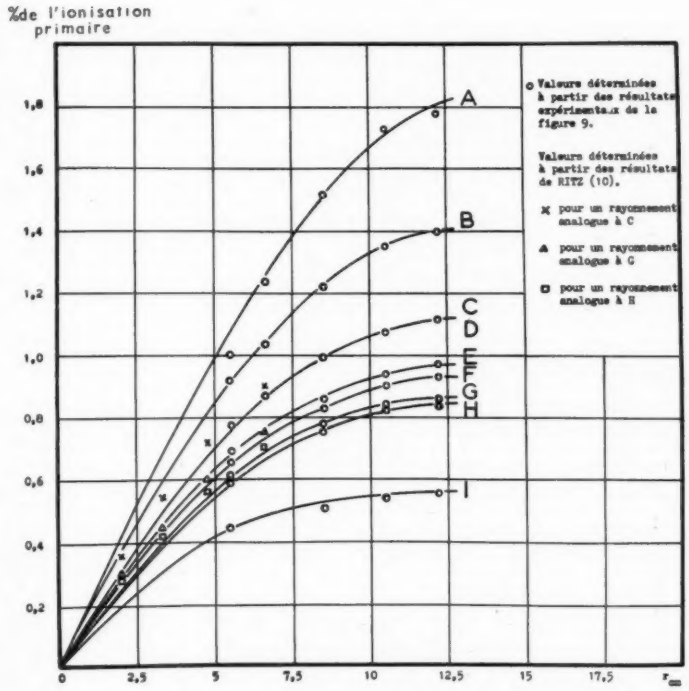


Fig. 10. Contribution des photons diffusés dans un cylindre de rayon r centré sur un faisceau de rayons roentgen de diamètre nul.

fonction de l'épaisseur de ce dernier (Fig. 9). L'addition des tuyaux de téréphtalate revient à augmenter le volume d'air autour du faisceau, l'échelle des abscisses de Fig. 9 pouvant être convertie en épaisseurs d'air en utilisant le fait que 1 mg/cm² de téréphtalate est équivalent à 0,71 mg/cm² d'air (P63).

Cette courbe extrapolée jusqu'à un diamètre de faisceau nul donne la valeur de la contribution des photons diffusés dans tout le volume de la chambre. Il est nécessaire d'introduire une très légère correction pour tenir compte du fait que l'émission électronique du téréphtalate est inférieure à celle de l'air. Nous avons comparé ces résultats corrigés avec ceux calculés pour notre chambre (qui peut être assimilée à un volume de rayon 5,5 cm) à partir des résultats de RITZ (10).

A partir des courbes de Fig. 9 et des résultats de RITZ, nous pouvons déduire les courbes donnant le pourcentage de rayonnement diffusé dans un cylindre de rayon donné centré sur le faisceau (Fig. 10). Nous avons placé sur ces courbes les résultats obtenus par RITZ (10).

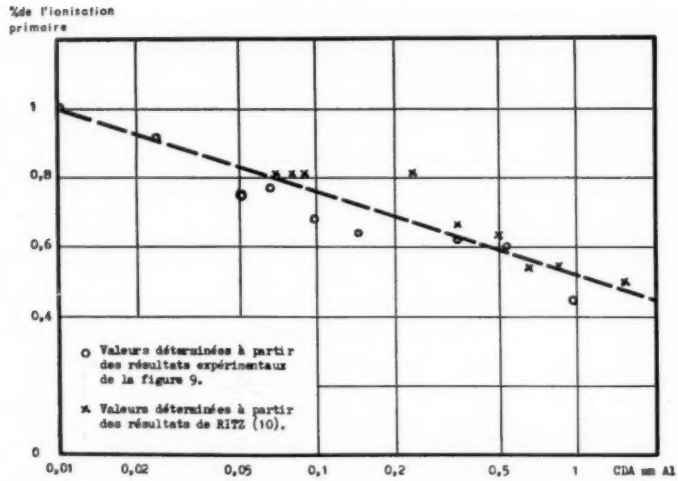


Fig. 11. Contribution des photons diffusés dans le volume de la chambre d'étude en fonction de la CDA du rayonnement.

Nous avons également tracé (Fig. 11) la courbe donnant la valeur de la contribution des photons diffusés dans le volume de la chambre en fonction du logarithme de la CDA du rayonnement utilisé. Les résultats de RITZ (10) ont été portés sur le graphique et l'accord est satisfaisant.

D. Saturation

L'étude de la saturation a été faite dans les conditions suivantes : 40 kV, 2 mA, filtration additionnelle nulle, la chambre étant placée le plus près possible du tube à rayons roentgen de façon à obtenir un débit important.

Pour une dose d'exposition de 126 r/min dans le plan du diaphragme d'entrée de la chambre, la saturation est obtenue pour 4 500 volts. En effet, l'extrapolation de la courbe $1/i$ en fonction de $1/V$ nous donne une variation du courant d'ionisation de $1{,}4^{0}/_{00}$ lorsque V varie de 4 500 à 9 000 volts ou de 5 000 à 10 000 volts (16).

Ce travail a été effectué au Laboratoire de Dosimétrie du Service Central de Protection contre les Rayonnements Ionisants, dans le cadre de recherches sur les étalons de mesure des doses d'exposition. Il a pu être réalisé grâce à Monsieur le Professeur BUGNARD et Monsieur J. MASSIOT; nous désirons leur exprimer ici notre vive gratitude.

RÉSUMÉ

Les auteurs décrivent une chambre d'ionisation à plaques parallèles d'un type spécial qui a été utilisée dans les études sur les étalons français de mesure de dose d'exposition. Cette nouvelle chambre permet la mesure de l'ionisation lorsque la longueur de l'électrode de mesure

ter vers zéro. Le dispositif décrit a permis de mesurer les divers facteurs de correction utilisés da la détermination absolue des doses d'exposition, en particulier: l'atténuation de l'air, les déformations du champ électrique, l'influence des différences de potentiel de contact ainsi que la contribution des photons diffusés.

SUMMARY

A special type of free air ionization chamber is described; it was used for investigations on the French exposure dose standard. This new chamber enables ionization measurements to be made when the length of the measuring electrode tends toward zero. The various correction factors used in the absolute determination of exposure doses were measured using the chamber described, especially air attenuation, electric field distortions, influence of contact potential and scattered photons.

ZUSAMMENFASSUNG

Ein besonderer Typus einer Freiluftjonisationskammer wird beschrieben. Sie ist zur Untersuchung des französischen Bestrahlungsdosisstandards benutzt worden. Diese neue Kammer ermöglicht Jonisationsmessungen durchzuführen, wenn die Länge der Messelektrode nahezu Null ist. Die verschiedenen Korrektionsfaktoren, die man bei der absoluten Bestimmung der Bestrahlungsdosis benutzt, wurden mit Hilfe der beschriebenen Kammern gemessen, insbesondere die Luftschwächung, die Störungen des elektrischen Feldes, der Einfluss des Kontaktpotentiales und der Streuphotonen.

BIBLIOGRAPHIE

1. Allisy, A., Delavergne, L. and Wyckoff, H. O.: Intercomparison of French and U. S. roentgen ray standards. Acta radiol. 48 (1957), 484.
2. Bunde, E., Lang, D., Pohlit, W. und Sewkor, A.: Absolute Dosismessung im Gebiet der weichen und sehr weichen Röntgenstrahlung mit Hilfe einer totalabsorbierenden Ionisationskammer. Z. Naturforsch. 9a (1954), 129.
3. Day, F. H. and Taylor, L. S.: Absorption of X-rays in air. J. Res. NBS 40 (1948), 393.
4. Hubner, W.: Communication personnelle.
5. Jaeger, R.: Wahrer und scheinbarer Schwächungskoeffizient inhomogener Röntgenstrahlung. Physik. Z. 36 (1935), 41.
6. — Zur Standard-Dosimetrie der Grenz- und Weichstrahlen. Z. angew. Physik 6 (1954), 494.
7. Jennings, W. A.: Beryllium-window tube outputs; further considerations regarding the use of constant and pulsating tension. Acta radiol. 6 (1951), 477.
8. Oosterkamp, W. J. and Proper, J.: Free-air and thimble ionization chambers for Grenz-ray dosimetry. Acta radiol. 37 (1952), 33.
9. Rajewsky, B., Bunde, E., Dormeich, M. et coll: Darstellung, Wahrung und Übertragung der Einheit der Dosis für Röntgen- und Gammastrahlen mit Quantenenergien zwischen 3 keV und 500 keV. PTB (1955).
10. Ritz, V. H.: Design of free-air ionization chamber for the soft X-ray region (20—100 kV). Radiology 73 (1959), 911.
11. Somerwil, A.: A grid ionization chamber of special design for the measurement of soft radiations. Acta radiol. 37 (1952), 44.

12. TAYLOR, L. S. and STONEBURNER, C. F.: The measurement of low voltage X-ray intensities. J. Res. NBS 9 (1932), 769.
13. TROUT, E. D. and GAGER, R. M.: Physical characteristics of soft roentgen rays. Amer. J. Roentgenol. 62 (1949), 91.
14. VENNART, J.: Some physical measurements in the grenz ray region. Brit. J. Radiol. 27 (1954), 524.
15. WYCKOFF, H. O. and KIRN, F. S.: Standard chambers requirements for 250 to 500 kV X-rays. J. Res. NBS 58 (1957), 111.
16. — and ATTIX, F. H.: Design of free-air ionization chambers. NBS Handbook 64 (1957).
17. ZIELER, E.: Zur Charakterisierung weicher Röntgenstrahlung durch Halbwertschicht und Homogenitätsgrad. Strahlentherapie 93 (1954), 579.
18. — Dosismessungen an Berylliumfenster-Röhren für Spannungen von 10 . . . 100 kV. Strahlentherapie 100 (1956), 595.

REPORT OF THE

INTERNATIONAL COMMISSION ON RADIOLOGICAL UNITS AND MEASUREMENTS

Committee on the Methods of Evaluating Radiological Equipment and Materials Subcommittee on Focal Spots

Report of the Subcommittee IV.

1 — Method of Focal Spot Image Formation and Measurement

Introduction

At the 1956 meetings of the International Commission on Radiological Units and Measurements, a new committee was established to study methods of evaluating radiological equipment and materials; it operates under the Chairmanship of Dr. B. COMBEE of the Netherlands with Dr. E. D. TROUT of the U. S. A. as Vice-Chairman. A series of subcommittees have been set up to deal with specialized subjects. The report to follow is the first of the subcommittee reports to be completed.

As problems of radiation units and measurements have developed during recent years it has become increasingly evident that it is not feasible for the ICRU to publish a regular triennial report covering all areas of interest to the Commission. Therefore, it has been decided that as different committees or subcommittees of the Commission complete their reports they will be released for whatever Journal publication may appear to be appropriate. Approximately every three years the committee will endeavor to compile as many of these reports as possible into a single report. By this procedure it is hoped to avoid holding up the release of one completed study, simply to have it included in a single report with all other studies.

This report of Subcommittee IV of the ICRU is the first such report to be released in the manner described above. It will also be included as Appendix IV in the triennial report of the ICRU due for publication within a few months.

Committee IV of the ICRU consists of the following members:

B. COMBEE, Chairman, Netherlands, E. DALE TROUT (Vice Chairman), U. S. A., E. ZIELER (Technical Secretary), Germany, H. BERGER, Germany, BERNARD O'LOUGHLIN, U. S. A., A. S. JOHNSTONE, England, J. MASSIOT, France, D. J. STEVENS, Australia, S. W. SMITH, U. S. A., C. W. WEGELIUS, Sweden.

The Subcommittee (IV—1) that prepared this specific report consists of the following members.

T. ROGERS (Chairman), U. S. A., G. M. ARDRAN, England, E. FENNER, Germany, R. GRIFFOUL, France, A. KUNTKE, Germany.

This report has been reviewed and approved by the ICRU.

LAURISTON S. TAYLOR

Chairman International Commission on Radiological Units and Measurements

The task of this Subcommittee has been considerably simplified by the instruction given to it in the report of ICRU IV dated August 14, 1958, as follows:

"Only the tools for making an accurate measurement under any desired operating conditions should be specified. The specification of tolerances allowable from nominal focal sizes, of the tube current and voltage conditions should be considered to be outside the scope of ICRU IV."

Consistent with this instruction, the Subcommittee has given special attention to methods of measurement of focal spot size in X-ray tubes for diagnosis. In this report we submit our recommendations for a measurement method and procedure suitable for such tubes, particularly of the line focus variety, when operated at voltages not exceeding 150 kvp.

At the ICRU meeting held in Geneva, September, 1958, it was suggested that Subcommittee 1 should also include in its task the method of measurement of uniformity of focal spots and that the studies should not be limited to diagnostic tubes but should include therapy tubes and possibly radioactive sources. The Subcommittee has not as yet been able to evaluate this suggestion. It is of the opinion, however, that a method applicable specifically for diagnostic tubes can and should be recommended at this time, and that therapy tubes and radioactive sources should be the subject of further deliberation.

The following recommendations represent a composite of the positions taken by the individual members of the Subcommittee with respect to the various points involved rather than the unanimous opinion of all. However, a majority are in agreement that the method proposed will give a substantially accurate measure of the effective focal size of an X-ray tube under the operating conditions obtained at the time the measurement is made.

RECOMMENDATIONS

Method of Focal Spot Image Formation and Measurement
(For diagnostic tubes at voltages up to 150 kvp)

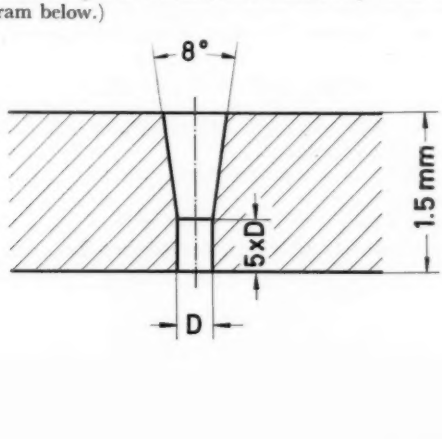
1. *Basic method.* Pinhole radiograph of focal spot, produced by pinhole accurately aligned with respect to "central ray" of X-ray beam.

2. *Details of pinhole camera*

a. Pinhole diameter:

0.030 mm for focal sizes below 1.0 mm
0.075 mm for focal sizes 1.0 mm to 2.5 mm
0.100 mm for focal sizes above 2.5 mm.

b. Diaphragm thickness, material, and pinhole profile: The pinhole diaphragm shall be manufactured from a 90/10 gold-platinum alloy, 1.5 mm thick. The cylindrical part of the pinhole shall be five times as long as its diameter and followed by a conical part with a divergent angle of 8°. (See diagram below.)



c. Focal spot to pinhole distance: 10 cm minimum (where focus location is not precisely known or directly measurable, precise location stereoscopically with dual pinholes is recommended).

d. Enlargement factor: 2.0 minimum, for focal sizes up to 2.5 mm, 1.0 minimum, for focal sizes above 2.5 mm.

3. *Photographic technique*

a. Type of film: any commonly used dental film (single coating).

b. Image density: 0.6 to 1.0, as measured in the most dense portion of the image. Density to be controlled by means of standard developing technique and proper exposure time at tube current and voltage factors employed. (This element involves a point of special difficulty, because of variations in structure of respective focal spots. If lines in focal spot are superimposed, greater maximum density results than in cases where lines are not superimposed, for equal exposure values. Hence a wide latitude in allowable maximum density becomes necessary.)

4. *Measurement of image*

a. Lighting: Back-lighted at approximately 20 foot-candles.

b. Measurement procedure: Use scaled magnifier with 0.1 mm scale divisions, 5 to 10 times magnification. Measure length and width, respectively, including all perceptible portions of the image, to nearest 0.1 mm. For irregularly shaped (non-rectangular) focal spots, measurement should be taken of all significant dimensions.

c. Correction of image measurements: All image dimensions shall be divided by the enlargement factor, accurately determined (see 2 d).

5. *Statement of dimensions*

a. Width of focal spot shall be stated as the width of the image divided by the enlargement factor.

b. Length of focal spot shall be stated as the length of the image divided by the enlargement factor, further corrected by a multiplier of 0.7. (See appendix A for explanation.)

c. For non-rectangular focal spots, all significant dimensions, including maximum diameter, shall be stated as indicated by actual measurements divided by the enlargement factor.

APPENDIX A

The proposal of a fractional multiplier for correction of the measured image length arises from the long-recognized fact that the lengthwise distribution of energy in the focal spot of line-focus tube tends to be peaked at the center and diminishes gradually to zero at the ends. Hence, the effective length, based on its effect on either radiographic definition or loading capacity, cannot be stated as equal to the measured image length as initially corrected. Some further correction is necessary. This point has been exposed, and to some extent explored in the papers heretofore presented by Polansky and O'Connor (1954), Rogers (1956), Fenner and Jochim (1957), and Servais (1958). However, we do not feel that work done to date has been sufficiently comprehensive to establish a suitable correction factor, and the suggestion above of a multiplier of 0.7 is given primarily by way of example or as an approximate value rather than as a final recommendation. We would like to recommend that ICRU IV enlist the good offices of a suitable research organization to explore this matter quantitatively over the entire range of diagnostic tubes commercially produced, with the objective of establishing a proper formula for focal spot length.

APPENDIX B. Off-Focus Radiation

It must be recognized that the effect of any off-focus radiation on definition is not taken into account by these measurements, and unless off-focus radiation is suppressed the definition to be expected from the tube may be influenced by it.

REFERENCES

FENNER, E. und JOCHIM, H.: Bestimmung der Brennfleckgröße aus der geometrischen Unschärfe. Fortschr. Röntgenstr. 87 (1957), 109.

POLANSKY, D. and O'CONNOR, D. T.: X-ray focal spot measurement. Nondestructive Testing, 12 (1954), 1.

ROGERS, T. H.: Definition and measurement of the focal spot size of an X-ray tube. Presented, Eighth International Congress of Radiology, Mexico City, July 24, 1956.

SERVAIS, J.: Le contrôle des foyers d'émission dans les tubes radiogènes et relation entre la largeur de foyer et la netteté relative des images radiographiques. J. Radiol. Electrol. 39 (1958), 383.

BOOKS RECEIVED

We acknowledge with thanks under this heading books received for review, which we trust will be regarded as a sufficient mark of appreciation of the courtesy of the sender. The reviews of selected items will appear as soon as opportunity affords.

ADVANCES IN BIOLOGICAL AND MEDICAL PHYSICS. Vol. VII. Editors: Cornelius A. Tobias and John H. Lawrence. Academic Press, New York 1960.

AUFDERMAUER, MAX: Die Spondylosis cervicalis (Die Wirbelsäule in Forschung und Praxis, Band 17). Hippokrates-Verlag, Stuttgart 1960.

BENTZEN, NIELS: Urethrographic studies of prostatic tuberculosis. Thesis, University of Copenhagen 1960.

BIAGINI, CARISSIMO, BOMPIANI, CARLO & PALEANI VETTORI, PIERO G.: Le radiazioni di alta energia. Basi fisiche e biologiche delle applicazioni terapeutiche. Societa Editrice "Universo", Roma 1960.

BOHLIG, H., JACOB, G. & MÜLLER, H.: Die Asbestose der Lungen. Genese — Klinik — Röntgenologie. Georg Thieme, Stuttgart 1960.

(THE) CELL NUCLEUS. Proceedings of the 1959 Meeting at the University of Cambridge by the Faraday Society. Butterworth, London 1960.

CRONKITE, EUGENE P. & BOND, VICTOR P.: Radiation injury in man. Its chemical and biological basis. Pathogenesis and therapy. Charles C. Thomas, Springfield, Ill., 1960.

DAVIDSEN, H. GÖSTA: Atrial septal defect. An investigation into the natural history of congenital heart disease. Munksgaard, Copenhagen 1960.

DIAMOND, HENRY D.: Die interne Krebstherapie (The medical management of cancer). Deutsche Übersetzung von P. Obrecht und P. Berg. Georg Thieme, Stuttgart 1960.

DI RIENZO, S. & WEBER, H. H.: Radiologische Exploration des Bronchus. Georg Thieme, Stuttgart 1960.

ECOIFFIER, J.: La radiculographie lombaire dans la sciatique. Masson, Paris 1960.

ERGEBNISSE DER WIRBELSÄULENFORSCHUNG (Die Wirbelsäule in Forschung und Praxis, Band 15). Zusammengestellt von Herbert Junghanns. Hippokrates-Verlag, Stuttgart 1960.

ETTER, LEWIS E.: Glossary of words and phrases used in radiology and nuclear medicine. Charles C. Thomas, Springfield, Ill., 1960.

FELSON, BENJAMIN: Fundamentals of chest roentgenology. W. B. Saunders, Philadelphia 1960.

FRIMANN-DAHL, J.: Roentgen examinations in acute abdominal diseases. Second edition. Charles C. Thomas, Springfield, Ill., 1960.

HEALTH PHYSICS, Vol. I. Progress in nuclear energy (PNE) Series XII. Second International Conference on the peaceful uses of atomic energy. Editors: W. G. Marley and K. Z. Morgan. Pergamon Press, Oxford 1960.

HÖSLI, P. O.: Anomalien der Harnwege im Kindesalter und ihre chirurgische Behandlung. S. Karger, Basel 1960.

KAINDL, F., MANNHEIMER, E., PFLEGER-SCHWARZ, L. & THURNHER, B.: Lymphangiographie und Lymphadenographie der Extremitäten. Georg Thieme, Stuttgart 1960.

KLINISCHE NEURORADIOLOGIE. Herausgegeben von K. Decker. Georg Thieme, Stuttgart 1960.

KÜNZLER, RALPH & SCHAD, NIKOLAUS: Atlas der Angiokardiographie angeborener Herzfehler. Georg Thieme, Stuttgart 1960.

LOW-LEVEL IRRADIATION. Symposium. Editor: Austin M. Brues. American Association for the Advancement of Science, Washington, D. C. 1960.

MAISIN, HENRI: Contribution à l'étude du syndrome médullaire après irradiation. É ude expérimentale. Éditions Arscia, Bruxelles 1959.

MALLET, L.: La lumière bleue. J. B. Baillière et Fils, Paris 1960.

MECHANISMS IN RADIOBIOLOGY, Vol. II. Multicellular organisms. Editors: Maurice E rera and Arne Forssberg. Academic Press, New York 1960.

MEDICAL PHYSICS, Vol. III. Editor: Otto Glasser. The Year Book Publishers, Chicago 960.

MITCHELL, J. S.: Studies in radiotherapeutics. Harvard University Press, Cambridge, ! lass. 1960.

NÈGRE, A. & ROUQUET, F.: Précis de technique radiologique. 4e édition. G. Doin & Cie., Paris 1960.

PISANI, G., MALARSINA, A. & SAVINO, G.: Diagnostica radiologica del cancro della mamr ella. Minerva Medica, Torino 1960.

RAYONS X, RADIO-ISOTOPES ET E.E.G. DANS L'ÉPILEPSIE. Ouvrage publié par les soi s de H. Fischgold et H. Gastaut. Masson, Paris 1960.

RECUEIL PÉRIODIQUE ENCYCLOPÉDIE MÉDICO-CHIRURGICALE. 13—14 cahier spécialisé radic liagnostic. Tomes I et II. Éditions Techniques, Paris 1960.

REPORT OF COMMITTEE II ON PERMISSIBLE DOSE FOR INTERNAL RADIATION (1959). ICRP Publication 2. Pergamon Press, Oxford 1960.

RIESSBECK, KARL-HEINZ: Zur Methodik der Röntgenstrahlen-Behandlung bösartiger Geschwülste. Zweite, erweiterte und neubearbeitete Auflage. Georg Thieme, Stuttgart 1960.

RODRIGUES, DIRCEU: Broncografia con bario e metilcelulose. Observacoes no henem e estudo experimental. Dr Dirceu Rodrigues, Rua Almirante Gonçalves 1865, Parana, Curitiba 1960.

ROUX, HENRY: Notions de radiobiologie. Masson, Paris 1960.

SCHMIDT, HERMANN & FISCHER, ERICH: Die okzipitale Dysplasie. Georg Thieme, Stuttgart 1960.

SELMAN, JOSEPH: The basic physics of radiation therapy. Charles C. Thomas, Springfield, Ill., 1960.

SPIRKO, SISTER CHRISTINA: Radiologic records. Charles C. Thomas, Springfield, Ill., 1960.

TALAIRACH, J., DAVID, M., TOURNOUX, P., CORREDOR, H. & KVASINA, T.: Atlas d'anatomie stéréotaxique. Repérage radiologique indirect des noyaux gris centraux des régions mésencéphalo-sous-optique et hypothalamique de l'homme. Masson, Paris 1959.

TURANO, LUIGI & SALOMONI, ENZO: Fisioradiologia dell'esofago. Normale e patologica. Societa Editrice "Universo", Roma 1960.

WACKENHEIM, A.: Neuroradiologie. Radio-anatomie normale et pathologique du crâne. G. Doin & Cie., Paris 1960.

YEAR BOOK OF RADIOLOGY 1959—1960 Series. The Year Book Publishers, Chicago 1960.

ZIMMER, E. A.: Filmfehler in der Röntgenpraxis. Artéfacts et conséquences d'erreur de manipulation sur le cliché radiographique. Georg Thieme, Stuttgart 1960.